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HISTOPATHOLOGY OF THE BRAIN AND SPINAL CORD IN A CASE PRESENTING A POSTINFLUENZAL LETHARGIC ENCEPHALITIS SYNDROME

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IOWA CITY

The occurrence of small epidemic foci of epidemic encephalitis in Iowa during the spring of 1919 raises the question of a new disease entity that can reach its ultimate solution only by the publication of necropsy studies and careful pathologic examinations, bearing in mind always the toxic or infectious origin of the changes. Clinically it is not always possible to differentiate toxic ophthalmoplegia with lethargy from Heine-Medin's disease, or botulism with ophthalmic symptoms.

REPORT CF CASE

History.—Through the courtesy of Dr. C. P. Howard, professor of internal medicine, it is possible to present the case of F. T. (Clinical No. 6075), a man, aged 37, who in January, 1919, had an attack of influenza followed by pneumonia from which he apparently completely recovered. On March 25, he was admitted to the university hospital complaining of pain and weakness in the arms and legs. Following his admission he became progressively worse, developing a typical syndrome of lethargic encephalitis which included (1) sleeplessness followed by drowsiness with nocturnal delirium, that deepened into a stupor from which he could be roused to answer questions intelligently; (2) cranial nerve involvement shown by early transient diplopia, ptosis, mask-like facies and difficulty in swallowing; (3) spinal cord symptoms shown by a late loss of knee jerks, incontinence of urine and feces and rigidity of the neck. Death occurred April 10, 1919.

Necropsy.—The brain weighs 1,530 gm. The dura is free, thin, with congestion of the blood vessels. The superior longitudinal sinus contains no thrombus. On cutting the dura, a large amount of cerebrospinal fluid escapes. The pia-arachnoid over the base and convexity is thin and transparent, with congestion of the pial vessels. It strips with ease. The convolutions are well formed but slightly flattened. In the left frontal region there is a pin-point cortical hemorrhage. The basal blood vessels show no sclerosis and are moderately filled with blood.

Longitudinal section through the middle of the thalamus shows the gray and white matter well differentiated. The lateral ventricles are normal in size.

The ependyma is smooth and there are no cysts in the choroid plexus. The blood vessels of the cerebrum, especially in the centrum ovale and the basal ganglions, are congested and the lymph spaces widened. The pons and medulla show congestion and hyperemia.

The cerebellum shows no gross pathology.

The blood vessels of the spinal cord, especially those over the dorsolumbar region of the cord, are markedly congested, showing distinctly through the dura mater. On section, the cord shows brownish areas bordering the lateral portion of the horn and in the posterior columns symmetrically placed; and reddish or pinkish areas extending in radially from the periphery of the cord. The differentiation between the gray and white matter is not distinct, and the color is a dirty gray (Fig. 1).

Specimens from the brain and spinal cord were hardened in alcohol or in 10 per cent. formaldehyd and stained with thionin, Mallory's phosphotungstic acid hematoxylin, Herxheimer's fat stain, hematoxylin and eosin and osmic acid.

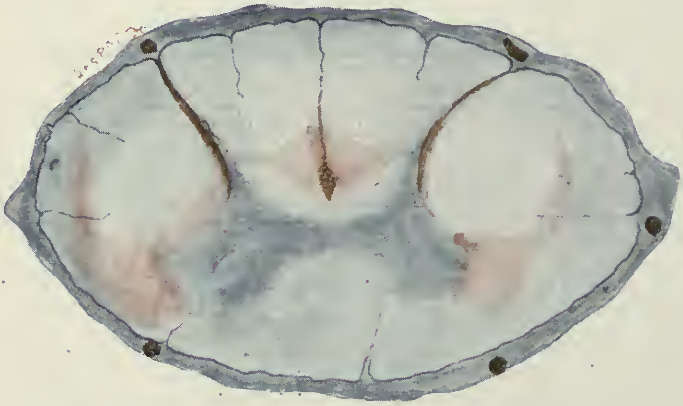

Fig. 1.—Dorsal region of the spinal cord showing a loss of differentiation between the gray and white substance, congestion of the pial blood vessels, and brownish areas of extravasated blood. Drawing from gross specimen, unstained; × 8.

Sections from different portions of the cerebral cortex stained with thionin show, especially in the frontal and anterior central convolutions, a disturbance of the cortical architectonics which is chiefly due to congestion of and infiltration about the blood vessels. There is an increase in the number of nuclei within the vessel walls, and a deposit of bluish staining granules. Yellowish pigment granules are also found either free in the vessel wall or deposited within epitheliod cells. With sudan III these granules are brilliant red. A few lymphocytes and plasma cells are present in the perivascular spaces. There is no new blood vessel formation. The marginal glia feltwork is denser beneath a slightly thickened pia-arachnoid, and there is an increase in the number of nuclei in the marginal zone. The nerve cells in the ganglion layers show a loss of the Nissl bodies. The cell outline is rounded, and the nucleus is often eccentric. The cytoplasm may show vacuolation or reticulation. Three to five satellite neuroglia cells are occasionally seen at the base of the large pyramidal cells.

Scattered irregularly through the cortex there are small, round, deeply staining masses of nuclear substance, which are about one third the size of a small neuroglia nucleus. In size and staining reaction they suggest neuronophages, but in their distribution they bear no relationship to nerve cells. A few rod cells are present (Fig. 2).

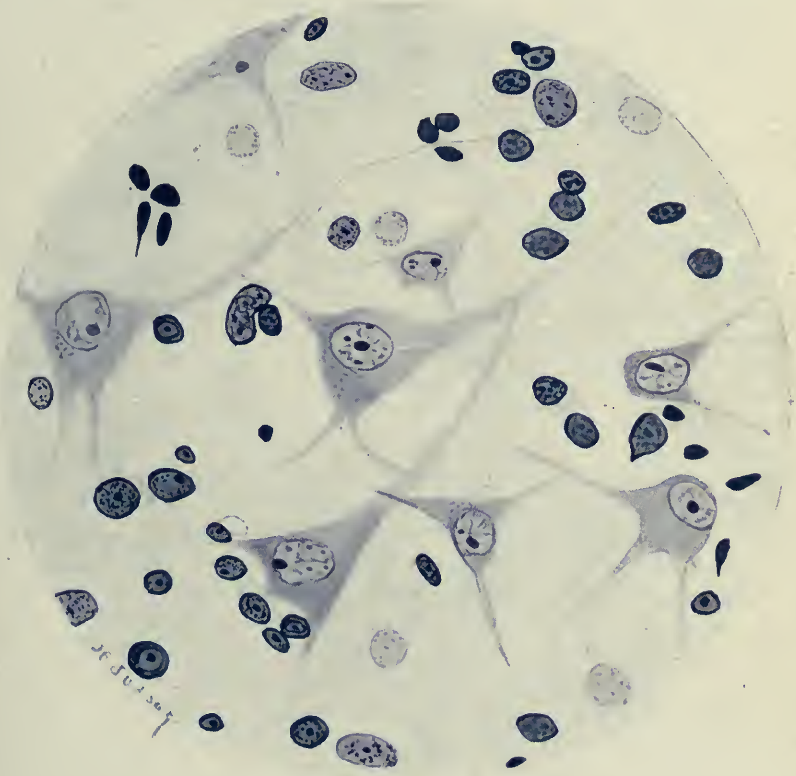

Fig. 2.—Section from the cortex showing nerve cell changes, satellite neuroglia, two types of neuroglia cells and the small, deeply staining cells which may be Maximow's polyblasts. Stain, thionin. Zeiss obj. D D; oc. 4.

In the white substance there is a deposit of granules in the vessel walls and some round nuclei in the perivascular spaces. One section shows a miliary hemorrhage at the cortical margin.

Sections through the basal ganglions show the blood vessels in the gray matter standing out sharply because of the presence of round, homogeneous, deeply staining bodies in the adventitia and in the perivascular spaces. There is also an increase in the cells in the vessel walls. Under the low power the homogeneous bodies give the appearance of a perivascular infiltration of lymphocytes and plasma cells, but under the high power only a few cells are found (Fig. 3).

The nerve cells have lost their angular outline, are rounded and stain a diffuse blue with thionin. A few of the ganglion cells contain yellow pigment granules, and about some of them there are from 5 to 7 neuroglia nuclei. The small, deeply staining glial forms predominate over the large, pale nuclei. (Fig. 4).

The upper part of the pons shows the blood vessels sharply outlined by an infiltration of mononuclear cells and homogeneous bodies in the adventitia and perivascular lymph spaces in the gray nuclei of the pons. There are a few granule cells filled with yellowish pigment granules in the infiltration. The blood vessels are congested. In the lower portion of the pons there is a proliferation of the subependymal glia with irregularities in the outline of the ependymal glia lining. The blood vessels are prominent, congested, and the nuclei in the floor of the fourth ventricle show deeply staining pigmented cells. In one portion there is an extravasation of blood into the perivascular space (Fig. 5).

The blood vessels of the cerebellum do not show the infiltration, collection of homogeneous bodies, and congestion noted in the basal ganglions, pons and medulla. The cells of Purkinje are rounded in outline, with a homogeneous cytoplasm and a loss of almost all of the Nissl granules. At times the cytoplasm is reticulated. The nuclear membrane may be irregular in outline and the nucleolus stains very deeply. The cell processes may show more distinctly than normal (Fig. 6).

Sections from the cervical and upper dorsal regions of the spinal cord show a proliferation of the ependymal cells about the central canal, with a blocking and closure of the lumen. The glial septums are thickened as a result of both congestion of the vessels within the septums and a neuroglia proliferation. Along the lines of the septums are collections of homogeneous bodies and a few lymphocytes. In the white substance adjacent to the lateral horn there is an extravasation of red blood cells, some of which have disintegrated.

About the margin of the cord and scattered through the lateral columns there are spaces in the ground substance, where both the axon and myelin sheath have dropped out. These are the areolar placques, or sieve-like areas of an infiltrative myelitis. The spaces are sometimes filled with a pale staining, homogeneous substance. The ganglion cells in the anterior horn show chromatolysis, and the nucleus is displaced to one side, but there are no indications of neuronophagia, and no hemorrhages into the gray matter. The pia arachnoid is thickened and homogeneous in appearance, with few nuclei. The posterior roots show areolar plaques and many deeply staining homogeneous bodies. There are no secondary column degenerations.

A section of the lumbar cord shows the dura mater thickened and homogeneous, with a round cell infiltration about some of the blood vessels. The outer layer of the pia arachnoid is infiltrated with lymphocytes, while the inner layers are thickened, hyaline in appearance and closely adherent to the cord, especially in the region of the posterior roots. The posterior nerve roots show congestion of the blood vessels, and in the entrance zone there are sieve-like areas and many homogeneous bodies. There is proliferation of the ependymal cells with obliteration of the central canal (Fig. 7). The nuclei in the vessel walls, especially in the gray matter, are increased, and some of the endothelial cells show double nuclei. Small, deeply staining particles of nuclear matter resembling neuronophages are present in the anterior horn, but nowhere are they seen in definite relationship to the nerve cells. The ganglion cells show the same change seen at higher levels. At the top of one of the posterior horns there is a small collection of red blood cells. The glial septums are widened and in places hyaline in appearance, and there are a few red blood cells along some of the septums. There are areolar spaces and swollen myelin sheaths along the periphery of the cord and in the posterior columns. Some of the spaces are filled with a delicate, homogeneous substance staining a pale pink with eosin (Fig. 8).

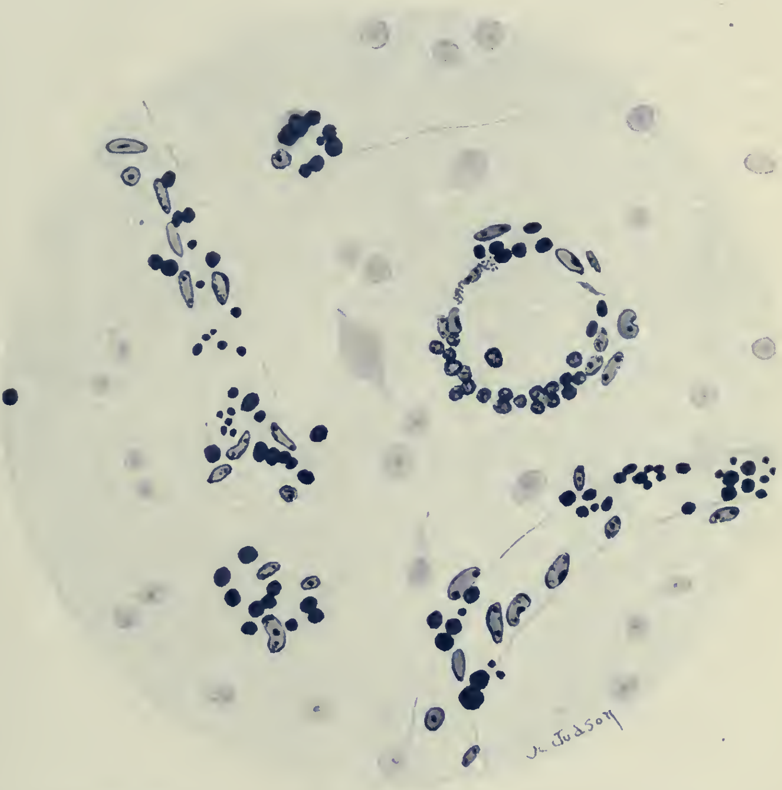

Fig. 3.—Perivascular infiltrations in the basal ganglions showing homogeneous bodies, endothelial cells and lymphocytes in the adventitial and perivascular spaces. Stain, thionin. Zeiss obj. D D; oc. 4.

Section through the cauda equina and the tip of the conus has a widened central canal, with proliferation of the ependymal lining and of the subependymal glia. The blood vessels about the central canal as well as those of the pia arachnoid and of the nerve trunks in the cauda are congested, but there is only slight infiltration (Fig. 9).

A section of a posterior root ganglion includes a large nerve trunk in which there is a calcareous deposit. There are also lime salt deposits in the connective tissue surrounding the nerve trunks. The ganglion cells show chromatolysis and loss of Nissl bodies.

Summary of Pathologic Changes.—This includes: Congestion and edema of both meninges and brain substance; acute encephalitis with perivascular infiltrations which are more marked in the basal ganglions and the nuclei of the pons and the medulla; nerve cell changes consisting of chromatolysis, cloudy swelling, and axonal reactions such as are found in the toxic states and with breaks in the continuity of the nerve fiber; alterations in the cells of Purkinje similar to those in fatigue and exhaustion; granular ependymitis of the fourth ventricle; acute diffuse infiltrative myelitis with extravasations of blood into the white substance; deposits of homogeneous bodies in the posterior roots, posterior root zones, and diffusely through the cord substance and perivascular collections of lymphocytes and plasma cells; closure of the central canal

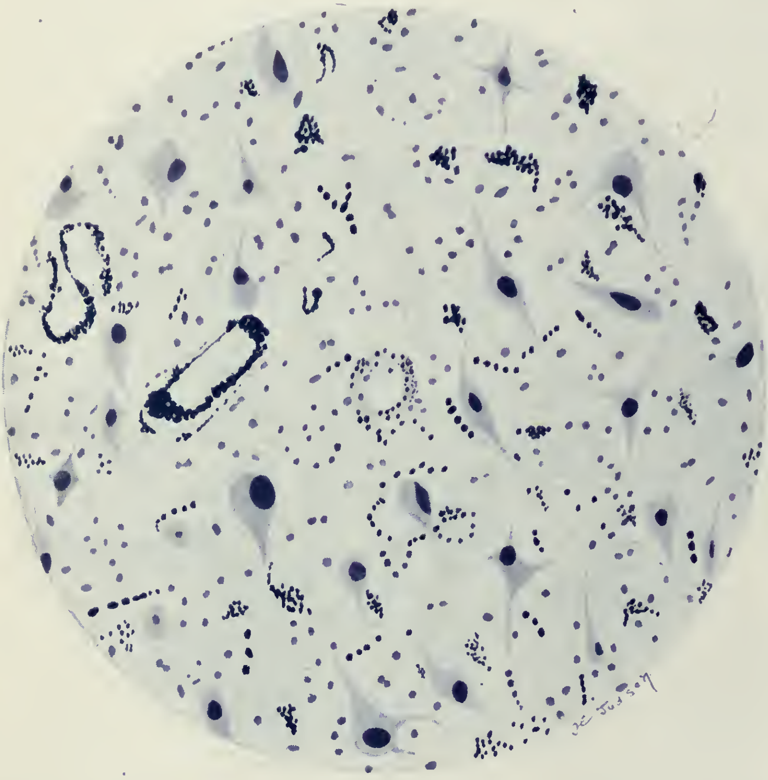

Fig. 4.—Section through the basal ganglions showing prominence of the blood vessels due to deposits in and about the vessel walls, nerve cell changes and focal collections of round cells and homogeneous bodies in the interstitial tissue. Stain, thionin. Zeiss obj. A; oc. 4.

throughout the entire cord, with widening at the tip of the conus medularis; nonsystemic areolar plaques; calcareous deposits in the posterior root ganglion; edema and thickening of the pia arachnoid with perivascular infiltrations of the pial blood vessels.

FINDINGS IN EPIDEMIC ENCEPHALITIS

The most constant findings reported in necropsy cases of epidemic encephalitis are congestion and edema of the brain and meninges. Microscopically, there is an infiltration of the adventitial and perivascular spaces with lymphocytes and plasma cells. These perivascular rings, occurring especially in the nuclei of the brain stem, have been considered almost pathognomonic. In the basal ganglion and the cranial nerve nuclei, the vessels stand out sharply differentiated from

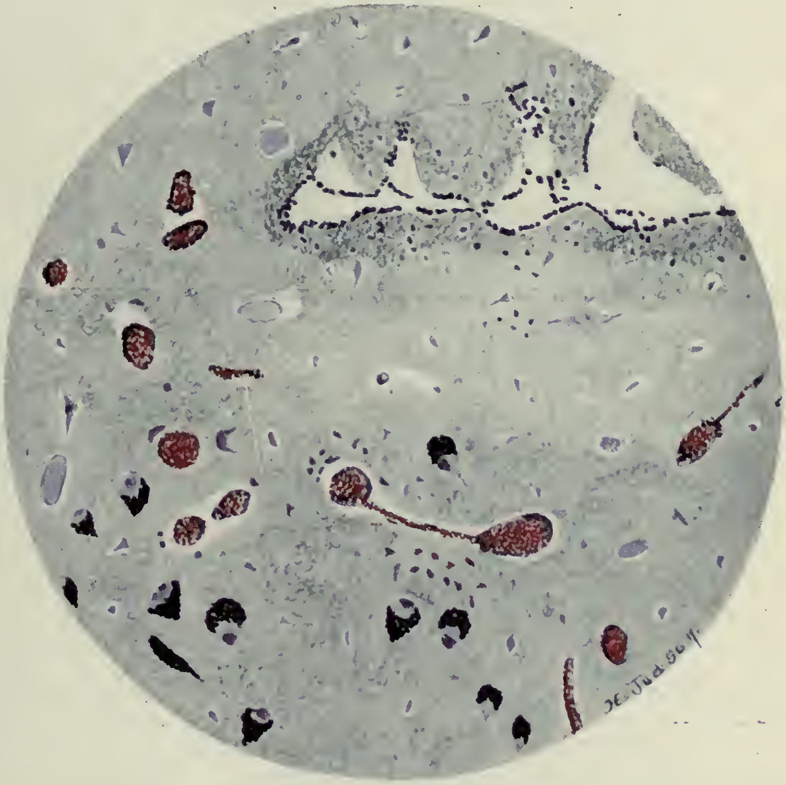

Fig. 5.—Section from the lower pons showing congestion of the blood vessels and granular ependymitis with proliferation of the subependymal glia. The nerve cells packed with pigment are part of the motor nucleus of the fifth cranial nerve. Drawn from a hematoxylin and eosin preparation. Zeiss obj. A; oc. 4.

the surrounding tissue by the ring of nuclei about a distended vessel lumen. The nuclei in the vessel walls show division forms and an increase in numbers, but there is no evidence of new vessel formation, and epithelioid cells either of vascular or glial origin are rarely seen. The neuroglia nuclei are moderately increased, but in no case is there a record of a double perivascular ring of which the inner circle represents the infiltrating cells and the outer the neuroglia nuclei gathering about the margin of the perivascular spaces. The character of the infiltrating cells varies. Bassoe and Hassen[1] report lymphocytes, plasma cells, polyblasts, fibroblasts and rod cells in the adventitial spaces, perivascular spaces and the adjacent parenchymatous tissue; Marinesco[2] finds plasma cells and lymphocytes with some vessels floating in a small pool of blood, and von Economo[3] shows the adventitial sheaths infiltrated with lymphocytes, leukocytes and plasma cells. The case of F. T. differs from those contained in other reports because of the slight cellular infiltration of the vessel walls, and the presence of masses of homogeneous bodies packed in the adventitia and in the perivascular spaces of the blood vessels of the cranial nuclei and the basal ganglions. These homogeneous bodies stain with thionin and cresyl violet. They are rounded and vary in size, and probably represent accumulations of degeneration products. Low and Mott[4] described them in a case of sleeping sickness due to trypanosomiasis.

In the spinal cord there are two forms of homogeneous bodies, one consisting of small, round, deeply staining masses found along the glial septums, in the posterior roots, the posterior root zones, and diffusely scattered in the white substance. They take a deep blue stain with hematoxylin, but do not stain with either osmic acid or sudan III. They have been reported as corpora amylacea or amyloid bodies (Marinesco) and were present in Breinl's[5] case of acute poliomyelitis. The other homogeneous bodies are found in the widened spaces in the ground substance, chiefly of the lateral columns. They take a definite pink stain with eosin and a pale stain with thionin, the effect being that of a faintly staining exudate. Stains for bacteria demonstrate no organisms within this homogeneous substance.

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[3] Von Economo, C.: Encephalitis Lethargica, Wien. klin. Wchnschr. **31**:850, 1918.

[4] Low, George C., and Mott, F. W.: Examination of the Tissues of a Case of Sleeping Sickness in a European, Brit. M. J. **1**:1666, 1889.

[5] Breinl, A.: Clinical, Pathological and Experimental Observations on the "Mysterious Disease." A Clinically Aberrant Form of Acute Poliomyelitis, Australian M. J. **1**:209 (March 16) 1918.

The nerve cell changes are more or less uniform throughout the brain and cord. There is chromatolysis and an axonal reaction found in the ganglion cells. The early axonal reactions shown by certain cells in the cortex are of especial interest in connection with the myelitic changes. The cytoplasm may be reticulated, and there are slight variations in the nuclear staining reactions. Neuronophagia has been reported in other cases, but is not present in the case of F. T. The small, deeply staining cells scattered through the cortex bear no

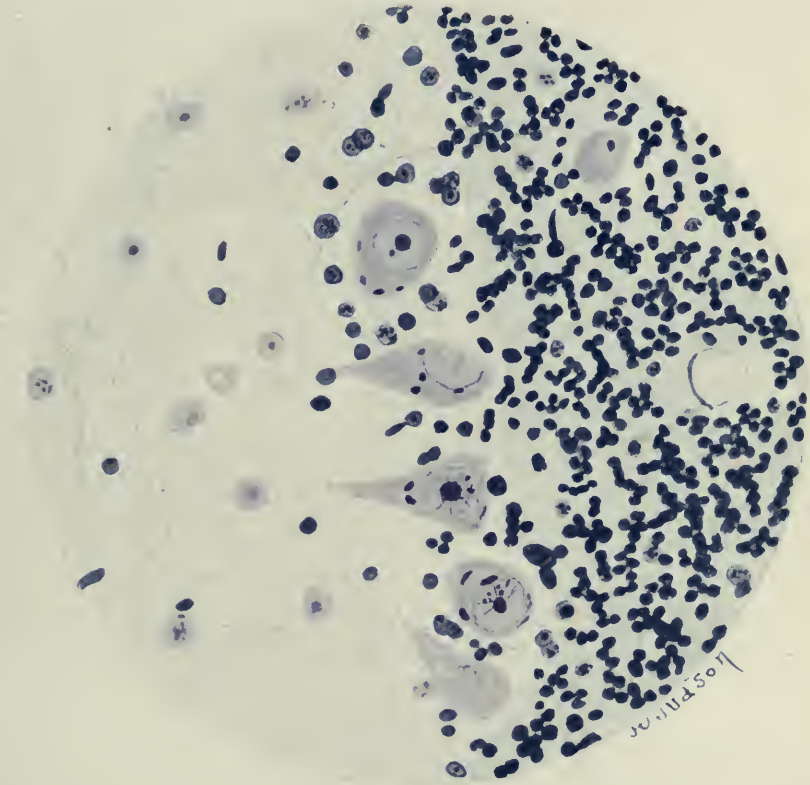

Fig. 6.—Section from cerebellum showing loss of Nissl bodies, chromatolysis and nuclear changes in the cells of Purkinje. Stain, thionin. Zeiss obj. D D; oc. 4.

relationship to the nerve cells. In both the cord and brain the ganglion cells show a satellite gliosis about the base of the cell, but in no instance are cells seen penetrating the nerve cell or within the nerve cell body. This, associated with the absence of hemorrhages into the nuclei and gray matter, makes an important pathologic differentiation from acute poliomyelitis of the Heine-Medin form. Marinesco emphasizes the constant finding in his cases of changes in the Purkinje cells in the cerebellum, which have already been described in this case. The picture is that seen in fatigue and shock.

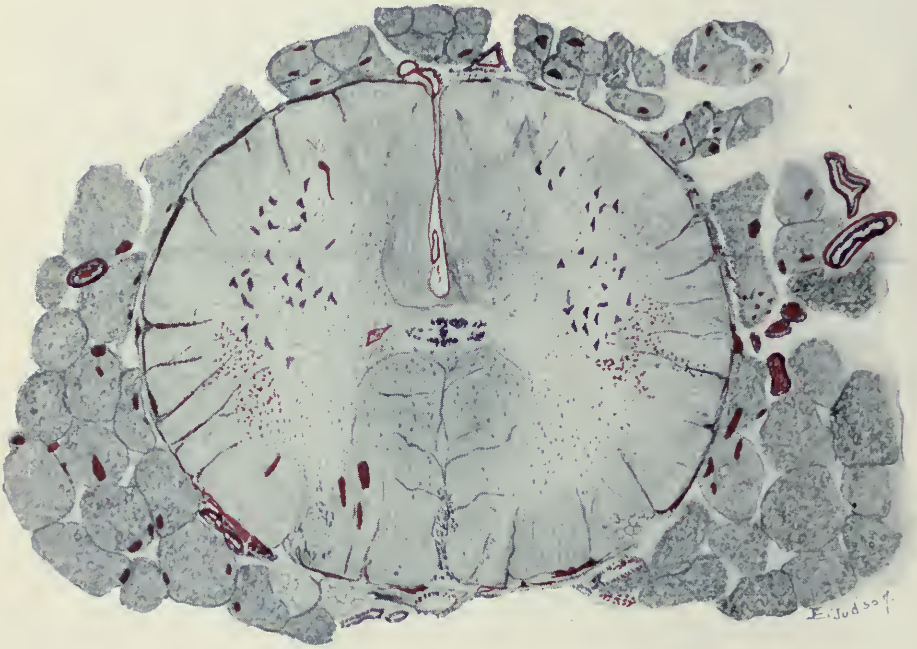

Fig. 7.—Section of lumbar cord showing closure of the central canal; areolar plaques along the anterior margin and in the posterior nerve roots; congestion of the blood vessels with slight hemorrhage into the white substance bordering the lateral horn; infiltrations in the lateral and posterior columns and subpial hemorrhages. Drawn from a hematoxylin and eosin preparation; × 8.

Spinal cord involvement was indicated by a loss of knee jerks and incontinence of urine and feces, other symptoms being masked by the coma. The diffuse nature of the process is shown by the dirty gray color of the cord with indefinite rusty discolorations in the white substance, which were present throughout the entire cord. Microscopically, the thickening of the neuroglia septums along which there are infiltrations of lymphocytes, plasma cells, and red blood corpuscles, the presence of an exudate in the spaces of the ground substance, congestion and perivascular infiltration, and the presence of areolar plaques and swollen myelin sheaths make a picture typical of that of an acute diffuse infiltrative myelitis. This picture is not new, Henneberg[6] giving a summary of the literature previous to 1911 on similar pathologic changes occurring after influenza. They are probably toxic in origin, the infiltration bearing a definite relation to the vessels arising from the vasocorona, the posterior and lateral vessels showing the greatest change. The thickening and slight infiltration of the pia-arachnoid of the cord presents a picture quite at variance with the severe, often purulent meningitis reported in cases of direct invasion of the meninges with B. influenzae.

No secondary degenerations are demonstrable by either Marchi or Weigert's myelin sheath stain. This is to be expected from the brief course and duration of the spinal cord involvement. The initial symptoms of pain may have been of root origin, judging by the collections of homogeneous bodies and the presence of areolar plaques in the posterior roots and posterior root zones.

The ventricular system, including the central canal, shows alterations. The ependymal lining of the fourth ventricle is broken and irregular, with proliferation of the subependymal glia. Granular ependymitis of this type, however, is not uncommon in cases coming to necropsy after 30 years of age. It is chronic rather than acute. The definite proliferation of ependymal cells about a closed central canal, the presence of a slight glial reaction, and the congestion of the neighboring blood vessels, together with the widening of the lower end of the central canal (Fig. 9), must be considered with reference to the theory that the virus of poliomyelitis is carried throughout the cord by way of the central canal, but the changes are so slight in the central gray matter, as compared to those in white cord mantle, that it seems improbable in this case for the spread to have occurred through this portion of the cord. No organisms can be demonstrated in the lumen. Bassoe and Hassin suggest that the amorphous mass in the closed central canal in their case may be made up of bacilli. Additional necropsy studies are reported by Pothier,[7] Wilson,[8] Bassoe,[9] Netter,[10] Buzzard,[11] McCaw and Stebbing,[12] Gordon,[13] Mott,[14] Marinesco[15] and Marie.[16]

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[6] Henneberg, R.: Die Myelitis: Handbuch der Neurology (Lewandowsky) **2**: Spezielle Neurologie I, 727, 1911.

ETIOLOGY OF LETHARGIC ENCEPHALITIS

Of the etiology there is little that can be said. Undoubtedly acute infections, especially pandemics such as the influenza in 1918-1919, are frequently followed by the lethargic encephalitis syndrome. Zuelzer[17] talks of the sleepy sickness in 1712, Longuet[18] reviews the Italian epidemic of nona, and Leichtenstern[19] gives a complete summary of the literature on the comatose form of influenza. The case of F. T. and others recently reported differ from the true influenzal encephalitis in the absence of thrombi, softenings, abscesses and organisms. In true influenzal encephalitis Pfeiffer's bacillus has been isolated from the areas of softening and hemorrhage, the cerebrospinal fluid and the meninges by Nauwerck,[20] Trouillet and Esprit,[21] and Pfuhl.[22] In the present epidemic of encephalitis no organisms have been found, if we except von Wiesner's[23] as yet unsubstantiated work, and no one has been able to transmit the disease to monkeys. It may be due to a toxin produced elsewhere in the body by the organism causing influenza, which may not be Pfeiffer's bacillus, or it may be due to an invasion by a new organism scattered to new localities by the world-wide movement of peoples. In the latter event the influenza pandemic by lowering resistance acts merely as a contributory factor. The same pathologic changes are found in trypanosomiasis and in the spirochete

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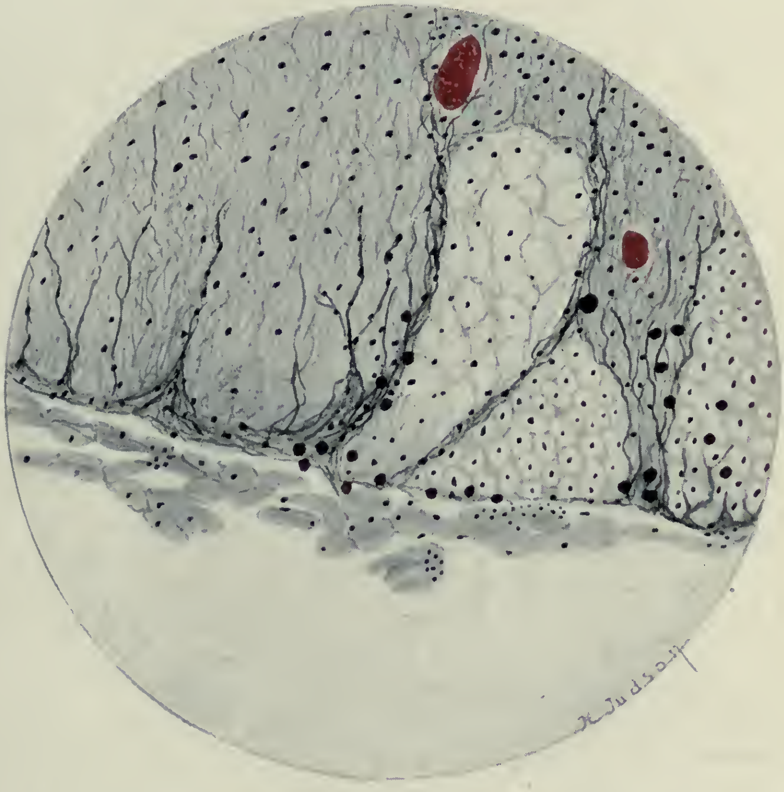

Fig. 8.—Section of cord showing a posterior root with areolar plaques, large purplish pink homogeneous bodies, small, deeply staining homogeneous bodies, thickenings of the pia-arachnoid and the marginal glia feltwork and congestion of the vessels in the cord. Drawn from a hematoxylin and eosin preparation. Zeiss obj. A; oc. 4.

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[23] Von Wiesner, R. R.: Die Aetiologie der Encephalitis lethargica, Wien. klin. Wchnschr. **30**:933, 1917.

infection of general paralysis; the changes differ only in degree and in location.

The combination of lethargy with cranial nerve involvement depends not on a specific virus, but on the location within the brain of the lesions produced by varying causes. Mauthner[24] localizes the sleep center in the floor of the fourth ventricle as a result of the findings in cases of nona, but MacNalty[25] explains lethargy and sleep as

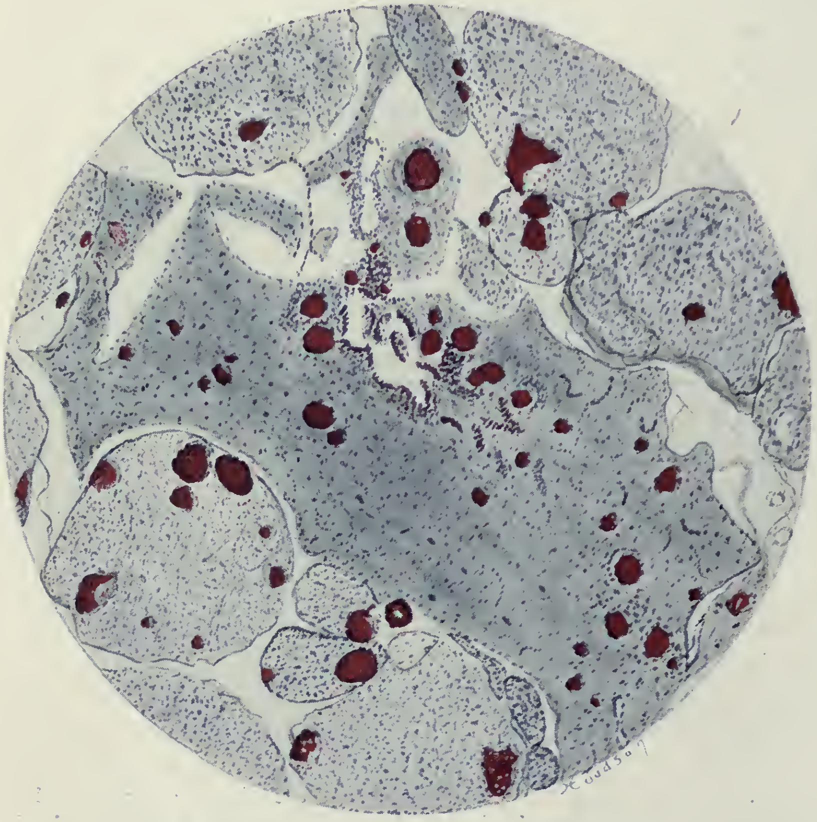

Fig. 9.—Section through the tip of the conus medullaris showing widening of the central canal, round cell infiltration, with congestion of the blood vessels of both the conus and the nerves of the cauda equina. Drawn from a hematoxylin and eosin preparation. Zeiss obj. A; oc. 2.

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[24] Mauthner, L.: Zur Pathologie und Physiologie des Schlafes nebst Bermerkungen über die "Nona," Wien. med. Wchnschr. **40**:961, 1890.

[25] MacNalty: Local Government Board Reports on Public Health and Medical Subjects. London. Report on an Inquiry Into an Obscure Disease, Encephalitis Lethargica, N. S. 121, 1918.

due, not to a special sleep control center; but to an interruption of the sensory stimuli on the way up to the cortex by lesions in the thalamic region. This shutting off of the outside world reproduces to a pathologic degree the mechanism of normal sleep. Undoubtedly the same syndrome is clinically given by some cases of acute polioencephalitis and botulism. The epidemic in Queensland and New South Wales summarized by Breinl[5] and the cases reported by Mills and Wilson[26] are undoubtedly poliomyelitis with the lesions in the basal ganglions and the cranial nerve nuclei. They are differentiated from this case and the cases variously reported as lethargic encephalitis, epidemic encephalitis, nona, X disease, and toxic or epidemic ophthalmoplegia with lethargy, by the presence of hemorrhages into the gray matter and marked neuronophagia, and by the fact that the virus is transmissible to monkeys in the same manner as that of poliomyelitis. There seems to be at the present time in various parts of the world epidemic foci of Heine-Medin's disease and of epidemic encephalitis which clinically present the common syndrome of ophthalmoplegia and cranial nerve involvements associated with a progressive lethargy; pathologically, they are two distinct processes.

The lethargic encephalitis syndrome should be considered in the same light as Landry's paralysis or the Brown-Séquard syndrome — that is, as a localizing syndrome resulting from varying causes. The pathologic findings of epidemic encephalitis indicate that we are dealing with a new disease for this country, but the relationship to the pathology in trypanosome infections and spirochetal infections must be kept in mind in the search for an etiologic agent.

CONCLUSIONS

1. The term lethargic encephalitis is the name of a clinical syndrome caused by lesions of varying types which are localized in the basal ganglions and the nuclei of the pons and medulla. It is not a disease entity.

2. Included in this syndrome there are cases of epidemic encephalitis, a disease new to this country, whose etiology is unknown, but whose pathology bears a close resemblance to that of African sleeping sickness.

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[26] Mills, C. K., and Wilson, George: Cerebello-Bulbar Polioencephalitis Originating During or After Epidemics of Influenza and of Poliomyelitis, Including the Record of a Case of Epidemic Encephalitis of the Lethargic Type, Arch. Neurol. & Psychiat. **1**:567 (May) 1919.

3. The case reported belongs to the new epidemic encephalitis group. The pathology is an acute infiltrative encephalomyelitis, the most marked changes occurring about the blood vessels of the thalamus, the cranial nerve nuclei, the floor of the fourth ventricle and in the white substance of the spinal cord.

A REVIEW OF THE EFFECTS OF GUNSHOT WOUNDS OF THE HEAD

BASED ON THE OBSERVATION OF TWO HUNDRED CASES AT U. S. GENERAL HOSPITAL NO. 11, CAPE MAY, N. J.*

CHARLES H. FRAZIER, M.D., AND SAMUEL D. INGHAM, M.D.
PHILADELPHIA

NEURCLOGIC ASPECTS

This report presents some general observations made in a series of 200 cases of wounds of the head, in practically all of which symptoms of injury to the brain was present. While the main purpose is to present a general survey of the cases as a group, certain aspects of the subject deserve more than casual mention.

Material.—These two hundred patients represent practically all of the cases of this type under observation at U. S. A. General Hospital No. 11, at Cape May, N. J., from October, 1918, to June, 1919. Of these patients, 88 per cent. suffered from wounds associated with demonstrable defects or fractures of the cranial bones, and 12 per cent. presented brain symptoms without demonstrable cranial injuries.

Patients with cranial defects	163
Patients with cranial fractures	13
No cranial injury evident	24

It will be seen that most of these patients presented cranial defects. These, with few exceptions, were the results of gunshot wounds of the head which were treated almost universally by early operation.

The associated brain injuries varied greatly, some being severe. In sixty-eight patients the cerebral symptoms were slight or could not be demonstrated when the patients were admitted to the hospital.

In 12 per cent. of the cases there was no definite history or evidence of cranial injury, but either general or focal symptoms of cerebral origin following traumatisms of the head seem to justify their inclusions in this series.

Symptomatology.—For the purpose of more systematic presentation, the manifestations of disturbed action of the nervous system have been classified into general and focal symptoms, in much the same sense that the symptoms caused by brain tumors are so classified; focal symptoms representing lesions of definite areas of the brain, and general symptoms those resulting from the effects of diffuse forces, such as concussion or pressure.

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* Read at the annual meeting of the American Neurological Association held in Atlantic City, N. J., June 17-18, 1919.

EARLY GENERAL SYMPTOMS

Among the early general symptoms of importance, according to the clinical records and the histories as given by the patients themselves, were disturbances of consciousness, amnesia, delirium and confusion, choked disk, slow pulse, headache and vertigo. While obviously incomplete, these histories, considered collectively, have a certain value.

Disturbances of Consciousness.—Data concerning the state of consciousness immediately following the injury was available in 132 cases. In twenty-two there was no loss, while in the remaining 110 immediate unconsciousness resulted from the injury which lasted from a few minutes to several weeks.

TABLE 1.—Period of Unconsciousness

	No. Cases
No loss	22
Less than one hour	51
One to twenty-four hours	21
One to six days	22
Over six days	16

Since there was such a wide variation in the manifestation of this symptom it is of interest to consider the factors active in its production. These include: the degree of concussion, or sudden force transmitted to the brain by the blow; the amount of brain tissue traumatized, and the secondary effects of the injury, including hemorrhage, edema and infection.

For the purpose of analysis these patients may be roughly divided into three groups: (1) those in whom there was no loss of consciousness; (2) those unconscious from a few minutes to twenty-four hours, and (3) those in whom this symptom was prolonged.

Group 1 included twenty-two patients with cranial defects who were not rendered unconscious by their wounds. Many of these had severe injuries, and fifteen exhibited permanent focal brain symptoms. It is well known that a blow on any part of the head may produce unconsciousness by concussion, but it is apparent that this factor was insufficient to cause this symptom in the patients of this group. In explanation it may be suggested that the force of the injury was apparently exerted over a small area, and even when the cranial bones were fractured, and the brain itself traumatized locally, the diffuse concussion must have been relatively slight. An illustration of this principle is furnished by the manner in which an egg may be broken, a quick, sharp blow producing a local fracture, while a slower but heavier blow results in extensive cracks in the shell. In the latter case the diffusion of the force is evidently greater than in the former.

Group 2 included seventy-two patients who were unconscious from a few minutes to twenty-four hours. It may fairly be assumed that cerebral concussion was the immediate cause of unconsciousness in this group, and that other factors were relatively unimportant in their effect on consciousness. Early surgical operations in many cases effected decompression, removed blood clots, pulped tissue, foreign bodies and bone fragments and controlled infection. It is probable that the character of the wounds themselves, in some instances, had the effect of automatic decompression, thus preventing prolonged unconsciousness.

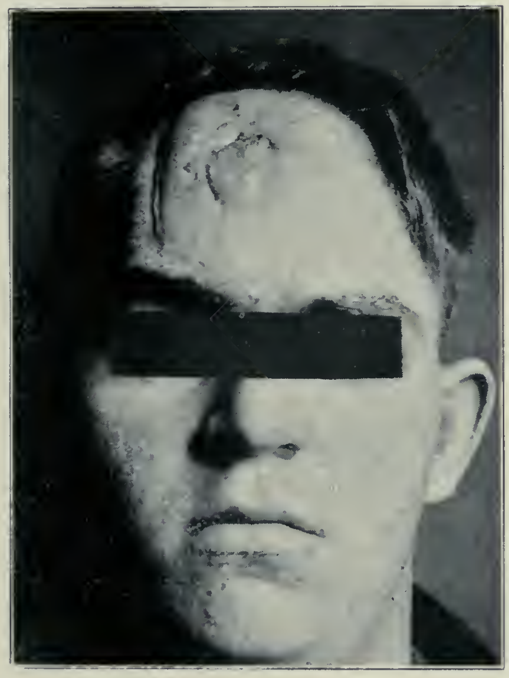

Fig. 1.—Patient with hernia at the site of the frontal defect.

Group 3 included thirty-eight patients who were unconscious for more than twenty-four hours. In many of these patients, in addition to cerebral concussion, it was evident that the secondary effects of trauma (hemorrhage, edema, infection) were important factors. Several of this group, with residual focal symptoms indicating severe brain injury, had had early operations in which the dura was not opened, hence decompression not effected. Some had deeply penetrating foreign bodies, and others severe wound infections and hernia cerebri. In this class were also included ten patients with cranial fractures not decompressed. Even from the fragmentary records available, the large proportion of injuries not relieved by decompression was striking in this group, injuries which must have produced severe secondary effects and high intracranial pressure. In none of the cases was there evidence that prolonged unconsciousness resulted from concussion alone. While it is at times difficult to differentiate cases of uncomplicated concussion from those in which intracranial hemorrhage and edema are also present, it is apparent that prolonged unconsciousness resulting from simple concussion is rare. On the other hand, conditions producing increased intracranial pressure, such as hemorrhage and edema not relieved by decompression, must be considered as important factors in prolonging the unconsciousness primarily induced by concussion in head injuries.

Incomplete loss of consciousness, dazed and stuporous states, delirium and mental confusion were common in the early histories, one or more of these conditions frequently following the period of unconsciousness, or replacing it as the immediate effect of the trauma. These symptoms were regarded as results of the same factors that caused unconsciousness, concussion standing in relation to the earlier, and the secondary effects of trauma to many of the more prolonged manifestations. In this connection it should be stated that definite symptoms apparently resulting from simple concussion occasionally persisted for several months.

Amnesia.—Amnesia was present in practically all of the patients exhibiting the symptoms mentioned, and the memory blank frequently antedated the injury. In two instances patients who were injured in France had no memory of having been out of the United States. Those who were dazed or delirious for a long time often retained a fragmentary or dream-like memory of isolated occurrences, or of their subjective mental processes at times fantastic and curiously related to actualities.

Headache, Vertigo, Choked Disk and Slow Pulse.—These symptoms were recorded with varying frequency, and were all more or less closely related to the secondary effects of injuries.

LATE GENERAL SYMPTOMS

When coming under observation in Hospital No. 11, two months or more after receiving head wounds, many patients still manifested cerebral symptoms of a general character. These included loss of

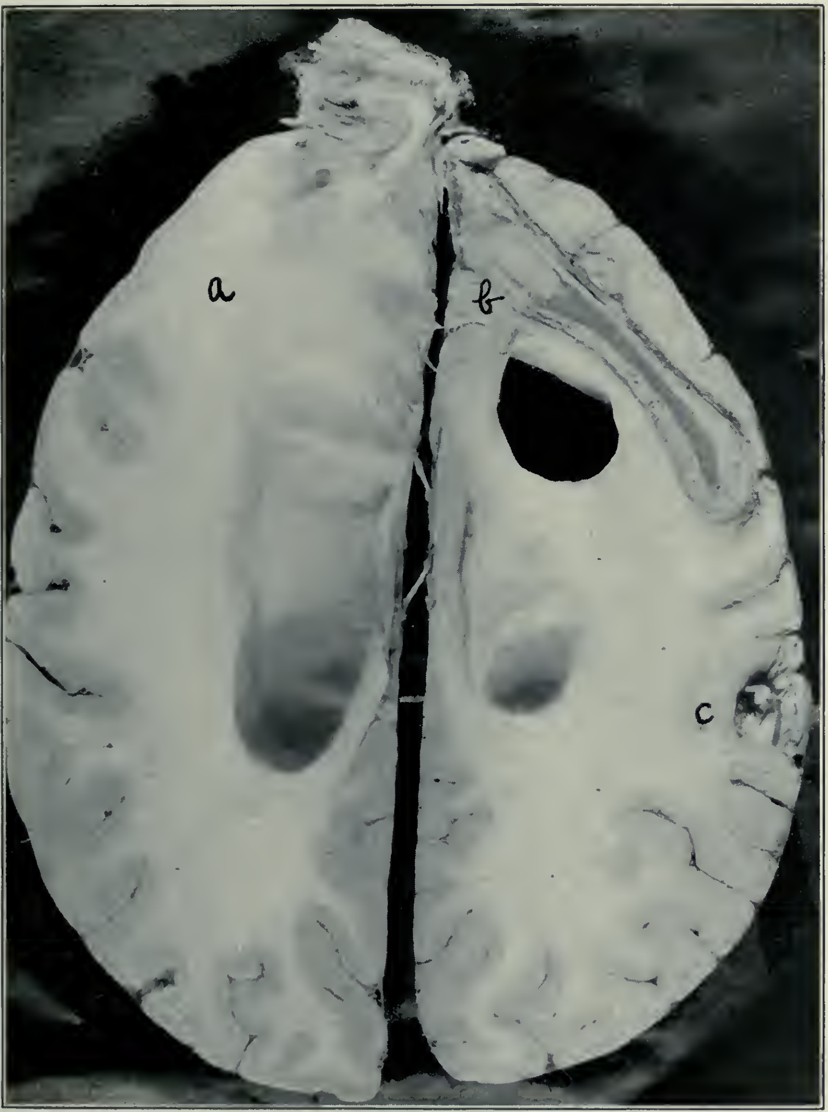

Fig. 2.—Brain showing (*a*) enlargement of left hemisphere and hernia cerebri at site of cerebral defect; (*b*) horn of dilated ventricle in relation with abscess cavity filled with inspissated pus; (*c*) bullet just beneath the cortex. Note relation of abscess cavity to trajectory between site of hernia and location of bullet.

memory, slow cerebration, indifference, mild depression, inability to concentrate, fatigability, nervous irritability, vasomotor and cardiac instability, general convulsions, fine tremors, irritable reflexes, headache, vertigo and restricted visual fields, but their manifestations varied in different patients as regards grouping, intensity and persistence. Some of them were present in most of the cases of severe head wounds, many of them were present in some of the cases, and exceptionally, a combination of these late general symptoms constituted the principal disability of the patient.

Almost without exception these symptoms diminished gradually, and ultimate recovery, apparently complete, occurred in from three to nine months after injury, where gross damage to the brain was absent. The tendency to recover from the symptoms, both general and focal, resulting from brain injuries of all degrees of severity, deserves special emphasis. Since it is fairly well established that regeneration does not occur in the central nervous system, it is evident that any nervous tissue may be affected to the extent of suspended function without suffering permanent damage, and recovery from the symptoms of brain lesions signifies returning function in injured but not devitalized neurons.

FACTORS CAUSING RESIDUAL GENERAL CEREBRAL SYMPTOMS

So far as could be determined, the following factors were operative in causing the late general symptoms in the series of cases under discussion:

1. Loss of cerebral tissue.
2. Injury to the brain without destruction of tissue.
3. Cranial defects.
4. Cicatrices.
5. Psychoneurosis.

Loss of Cerebral Tissue.—Symptoms resulting from the loss of cerebral tissue should properly be classified as focal, but these symptoms at times included intellectual impairment, or dementia, of which we have no definite knowledge in cerebral localization. Reference will be made, under the discussion of focal symptoms, to several instances in which the dementia apparently bore some relation to the location of the cerebral lesion.

Injuries to the Brain Without Destruction of Tissue. — These injuries include the effects of concussion and pressure, and also those of disturbed cerebral circulation and nutrition. Although, from the standpoint of pathology, changes of this nature are but imperfectly understood, it should be emphasized that they are common and impor-

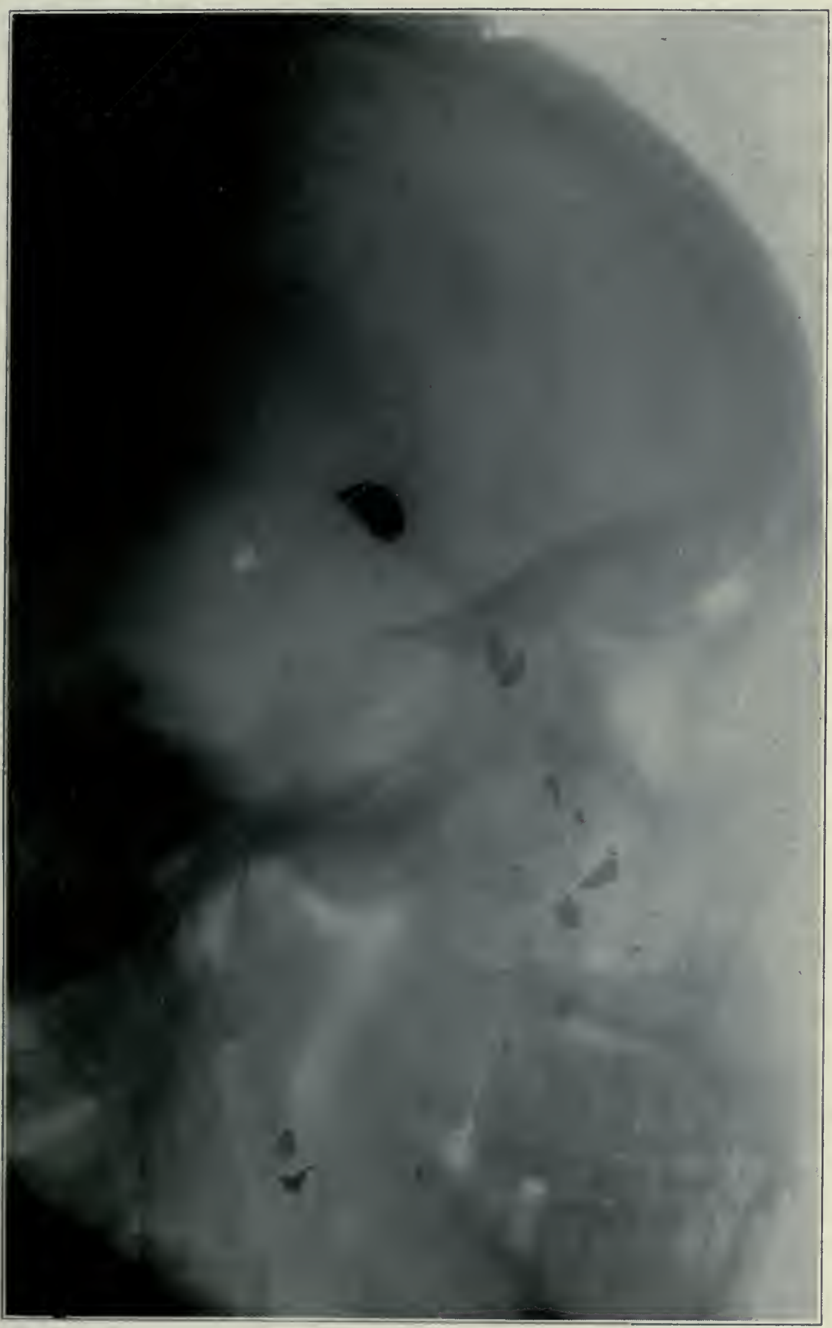

Fig. 3.—A "shower" of metallic fragments partly intracerebral and partly extracerebral.

tant. Most of the late general symptoms of head wounds are best explained on the basis of such disturbances. These symptoms include memory loss, slow cerebration, indifference, incapacity for sustained effort, vasomotor and cardiac instability, etc.

Cranial Defects.—These defects, particularly those large enough to permit fluctuation and pulsation, are commonly accompanied by vertigo, throbbing in the head and a feeling of insecurity, all of which are accentuated by active exercises and bending movements of the body. Headache, on the contrary, was noticeably unusual in the patients with cranial defects.

Cicatrices.—These sometimes act as irritating foci, causing nervous and reflex irritability, at times apparently precipitating general or focal convulsions. Headaches were often traced to pericranial and dural adhesions.

Psychoneurosis. — As an element in the symptomatology of this series this condition was comparatively unimportant. With three or four exceptions, anxiety and neurasthenic symptoms were present only to a degree commensurate with the nature of the injury. Conversion hysteria was not encountered in any of the cases.

Summarizing briefly the general cerebral symptoms resulting from wounds in relation to the etiologic factors, they may be divided into four groups: those due to (1) the immediate effects of the trauma; (2) the secondary effects of the trauma; (3) nondestructive injuries to cerebral tissue, and (4) destructive injuries to cerebral tissue. The first two of these groups of symptoms appear early, the latter two coming into prominence as the earlier symptoms subside.

The immediate manifestations consist mainly in disturbances of consciousness and in dazed, delirious and stuporous states, the principle causative factor being concussion.

The secondary effects of trauma (hemorrhage, edema, infection, etc.) add the symptoms of pressure to those of concussion.

Injuries to the brain tissue, not destructive in character, complicate all sorts of lesions, and cause symptoms which last for weeks or months, but which tend toward complete recovery. The syndrome of cerebral concussion (early disturbances of consciousness and prolonged mental symptoms including loss of memory, indifference, incapacity for sustained effort, mental slowness, etc.) probably has its pathologic basis in changes of this character.

VARIOUS SYMPTOMS

Transient Focal Symptoms.—Although the records were incomplete, they indicated that a considerable proportion of the patients suffered from focal symptoms of a transitory character, which disappeared completely or almost completely within one or two months following the injuries. Symptoms of this nature are to be explained by local injuries to the brain of a degree insufficient to cause tissue destruction.

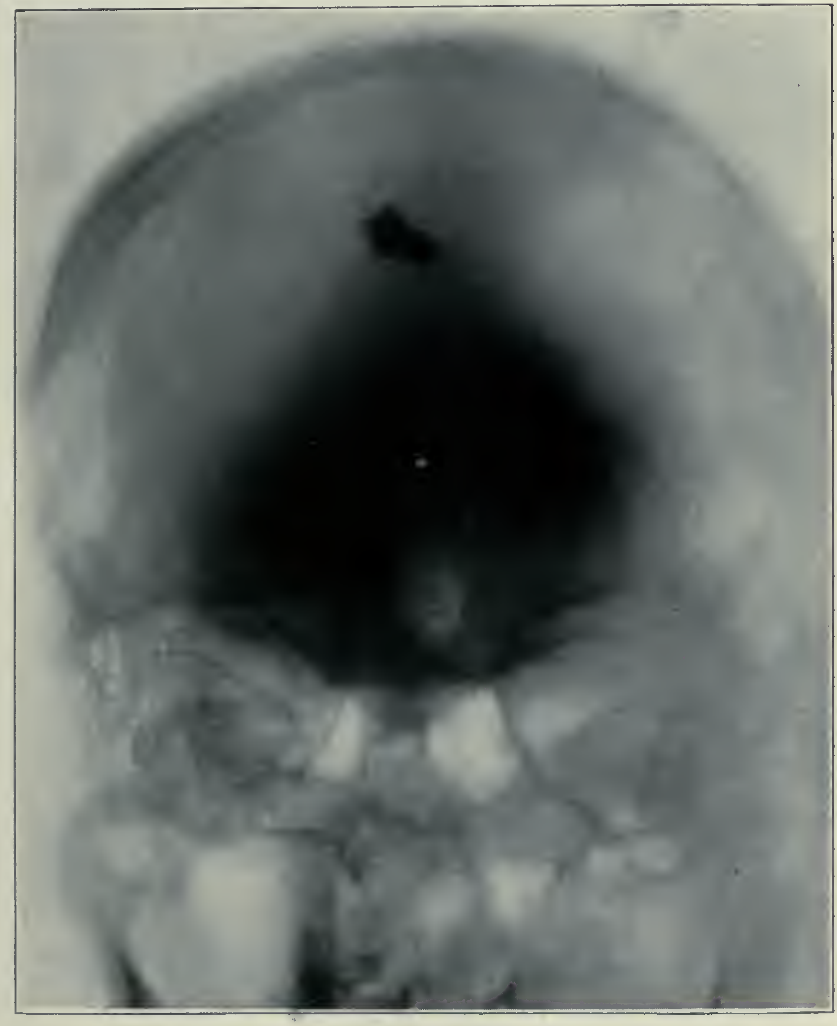

Fig. 4.—Large, single metallic fragment intrahemispheric.

Twenty-four patients gave histories of early hemiplegia which later disappeared entirely or left an insignificant remnant. In contrast, there were sixty patients with definite residual cerebral paralyses. Fourteen gave histories of aphasic disorders of a transitory character, while in sixteen some degree of aphasia persisted as a residual symptom. Four patients described symptoms evidently due to cerebellar disturbance, all of which recovered entirely. In no case was there evidence of a destructive wound of the cerebellum, a fact to be accounted for by the highly fatal nature of wounds involving the posterior cranial fossa.

Data concerning early sensory symptoms were for the most part unreliable as patients usually fail to note any but perceptual losses, and are even liable to confuse motor paralysis with anesthesia. In ten instances, however, there were fairly consistent histories of superficial anesthesia of unilateral distribution and of temporary duration.

Residual Focal Symptoms.—Under this heading are considered the focal symptoms which persisted while the patients were under observation, in most cases six months or more after the injury.

Motor Symptoms.—Of the entire series of 200, sixty, or 30 per cent., of the patients suffered permanent motor symptoms of cerebral origin. Of these, forty-three were hemiplegic, nine were monoplegic, and eight were paraplegic. The paralysis was of a severe degree in ten hemiplegics and three paraplegics, while in the remaining forty-seven the residual motor disability was comparatively slight when the patients were last examined.

A striking feature of these cases was the marked degree of recovery which invariably occurred. Probably without exception the patients were, immediately following the injury, completely paralyzed in the limbs affected. Twenty of them were admitted to the Cape May Hospital as litter patients; but when last examined they were all ambulatory, and many of them had a very fair amount of function in the paralyzed limbs. Notwithstanding this improvement there remained, in patients having destructive lesions in the motor areas, an irreducible minimum of paralysis.

The residual motor disabilities consisted of disturbances of voluntary motion of the arms and legs, and to a slight degree, of the face. Complete paralysis of a limb was never permanent. The functions of motility most disturbed were those of highly specialized and intricate character. Individual finger movements were uniformly most affected; finger flexion invariably returned in some measure, but extension was weaker and in two cases failed to reappear at all. All movements involving bilaterial groups of muscles were normal or showed insignificant disturbances.

Exaggeration of the tendon reflexes and hypertonicity of the muscles of the affected limbs was the invariable rule, although there was considerable variation in the degree of these conditions. Articular relaxation or increased range of movement in the joints as compared to the normal side was occasionally noted, and was demonstrable by the greater latitude of movement on passive manipulation after overcoming the hypertonicity of the muscles.

Incoordination constituted a factor in the disability of many of the paralytics, especially those showing a large measure of improvement though actual muscular strength was very fair.

Residual Sensory Symptoms.—Permanent impairment of cutaneous sensory perceptions of touch, pain and temperature was found in only eight cases, and in none was it present as a complete hemianesthesia. On the other hand, thirty patients showed impairment of ability to localize sensory stimuli acurately, to recognize dual contacts, and to appreciate passive movement and position in the extremities. In the same patients there was disturbance of the stereognostic sense. The constant association of impaired sensory discrimination with astereognosis indicates that the latter condition may be considered as a manifestation of the former.

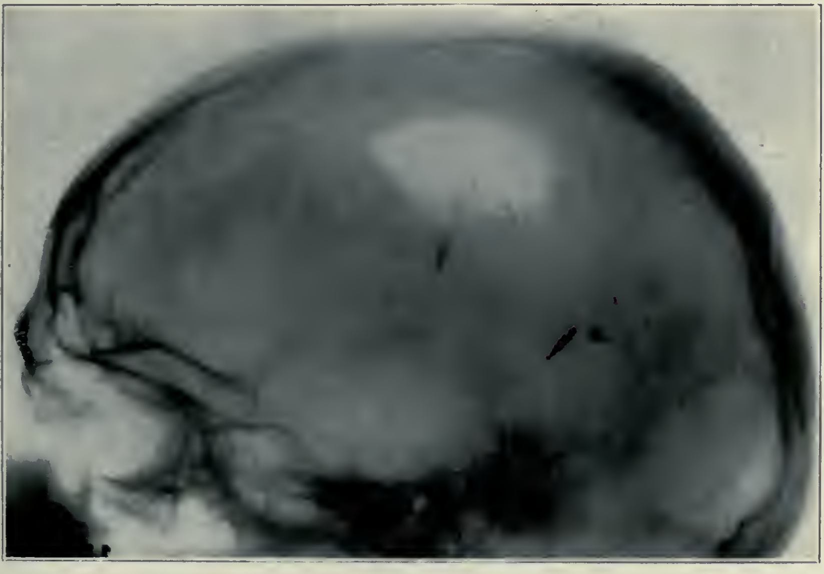

Fig. 5.—Three metallic fragments at a distance from the defect; two bone fragments within the margin of the defect.

Sensory and motor symptoms were frequently associated in the same case, and those having sensory symptoms almost invariably had motor impairment. The converse was not true, only 50 per cent. of the motor cases having demonstrable sensory symptoms. This relationship of motor and sensory symptoms may in part be explained by the dependence of normal movement, especially its coordination, on the discriminatory element of sensation.

Residual Aphasia.—In sixteen patients disturbances in the use of language remained six months or more after the wounds were received. Of these, ten were of the motor or dysarthric type, three of the sensory type with alexia as the most prominent symptom, and three were of the mixed type, manifesting disturbances both in the expression and in the interpretation of language. In none of the patients were the residual aphasic symptoms of severe degree, and all were able to carry on simple conversations fairly well. The patients with alexia were ultimately able to recognize letters and many words, but did not regain the ability to read understandingly to any practical extent.

Residual Visual Symptoms.—Cerebral wounds were associated with defects in the visual fields in eighteen cases, twelve of which were more or less complete homonymous hemianopsia, three were quadrant anopsias, two were symmetrical paracentral scotomas, and one was almost completely blind. Comparatively slight improvement was noted in the vision of these cases during the period of observation.

Mental Symptoms.—The occurrence of mental disturbance has been mentioned in connection with the general symptoms of cerebral injuries. Aside from the mental symptoms of cerebral concussion and the mild dementias of indeterminate type associated with many brain injuries, a few of the patients have shown late psychic symptoms which evidently resulted from cerebral wounds, and apparently bore some relation to the injured areas of the brain. This was true in four cases in which penetrating wounds involved both hemispheres. In three of these both frontal lobes were affected, and in the fourth a foreign body entered the right frontal region, penetrating to the left posterior parietal region near the cortex. Mental symptoms were pronounced in all of these patients and consisted of disorientation, loss of memory, emotional indifference and disregard of environment and personal appearance. In some measure they resembled the simple dementia of general paresis. Of the many patients with unilateral frontal lesions, some of them extensive, none showed characteristic psychic symptoms. These circumstances indicate the seriousness of bilateral brain lesions, and suggest the theory that either cerebral hemisphere may functionate in a way to minimize the effect of a lesion in the other.

Convulsions.—Convulsions occurred in twenty-eight patients, either before admission or while under observation at Cape May. In four of these the attacks were focal without general involvement, eleven had local spasms initiating general attacks, and in thirteen the convulsions were general so far as observations were recorded, although it is probable that some of these were preceded by unobserved focal symptoms. Attacks were observed in patients having lesions in the motor area and hemiplegia in which focal signs were definitely absent.

In three of the cases of this group it was found that attacks had occurred prior to military service, leaving twenty-five in which there was evidently a close relationship between the war wounds and the convulsions. In twenty-two of these the wounds involved the parietal region, and in twenty-one there was motor paralysis. In the remaining three cases the wounds were in the frontal, occipital and temporal regions. It is thus apparent that not only focal but general convulsions have been associated with motor areas of the brain in the great majority of the cases, and that irritation of these areas is more productive of general convulsions than of other parts of the cerebrum.

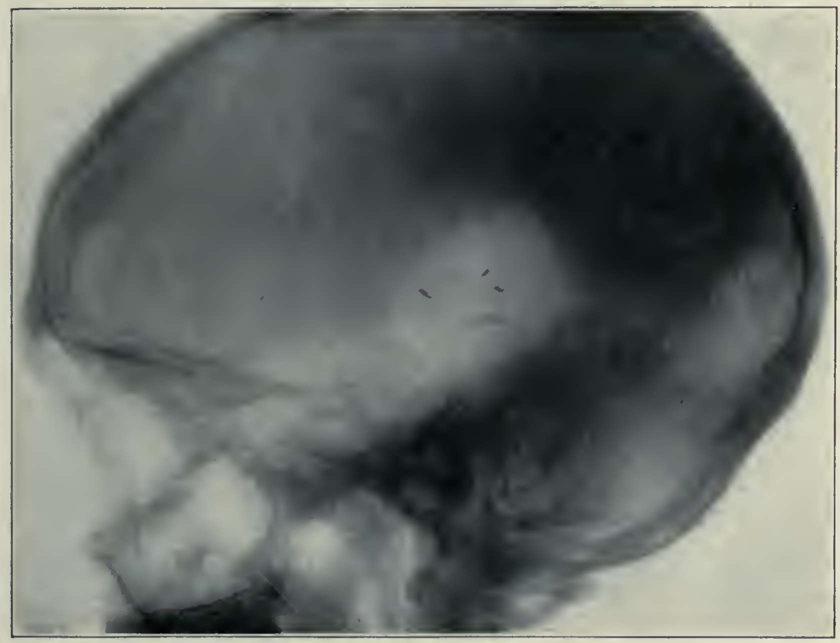

Fig. 6.—One minute bone fragment and three silver clips applied at operation overseas for control of pial hemorrhage.

Eighteen of the twenty-five patients have been free of attacks for several months, and three others have had but a single attack each. Four have had repeated convulsions over a prolonged period, thus evincing a tendency to chronic epilepsy; these were all hemiplegic, and the attacks were of the type which begin as focal convulsions, then become general with loss of consciousness.

The most frequent period for the occurrence of the attacks was soon after the wound had been received or after some operation on the head. About one half of this group of patients had isolated convulsions at such times without later recurrences.

It should be stated that, as a routine measure, bromids were given in 10 grain doses, three times a day, to all patients having convulsions, and to all those subjected to operations on the head, a measure which no doubt reduced the incidence of the attacks while the patients were under observation.

PATHOLOGY

The degree of injury to the brain varied from insignificant lesions to extensive losses of cerebral tissue. In twenty-three cases intracranial foreign bodies were demonstrated by the roentgen ray, some of them having almost traversed the cranial cavity. Small, indriven fragments of bone were common and were usually located in the vicinity of the cranial defect. In twenty-six cases the wounds were unhealed on admission, most of these having sinuses extending beneath the dura to fragments of dead bone or foreign bodies. One patient, who died four days after admission, had a large temperoparietal abscess and hernia cerebri. This patient was one of the two fatalities in the entire series of head wounds at the Cape May Hospital. The second fatality resulted from a complicating pneumonia and internal hydrocephalus, occurring after the wound had healed and the patient was convalescent.

During cranioplastic operations evidences of cerebral injury were at times noted. In such operations the dura was not usually opened, but occasionally it was necessary, and several times in this way cyst-like cavities filled with cerebrospinal fluid were exposed. In one notable case of this kind the operating surgeon, Major C. C. Coleman, opened such a cavity in the occipital lobe which communicated with the posterior horn of the lateral ventricle.

In estimating the area and extent of the cerebral lesions resulting from war wounds it may be stated as a rule that, in the absence of penetrating foreign bodies, the area of destruction of brain tissue conforms quite closely to the cranial defect, and extends but a few centimeters beneath the cortex. Foreign bodies may penetrate to almost any part of the cerebrum, even traversing the ventricles without causing death. The course of foreign bodies can be estimated by careful roentgen-ray studies, comparing their location with the wound of entrance.

TREATMENT

Besides the surgical treatment which is considered in another part of this report, special courses of treatment were given to practically all of the patients with the object of increasing their general efficiency, and of reducing to a minimum the effect of the disabilities from which they suffered. School, occupational and workshop courses were prescribed, according to conditions.

Patients with hemiplegia and paraplegia received daily treatments consisting of special massage, passive movements and electricity, also active exercises, employing the affected limbs to a maximum extent on gymnasium apparatus, and in recreational exercises in which handballs and footballs were found to be especially valuable. The results of this treatment were evident in reducing the spasticity and preventing contractures in paralyzed muscles and in procuring a maximum return of function. The training of the unaffected muscles to compensate as far as possible for those of impaired function gave the patients greater freedom of action, and the general poise, self-confidence and morale were noticeably improved.

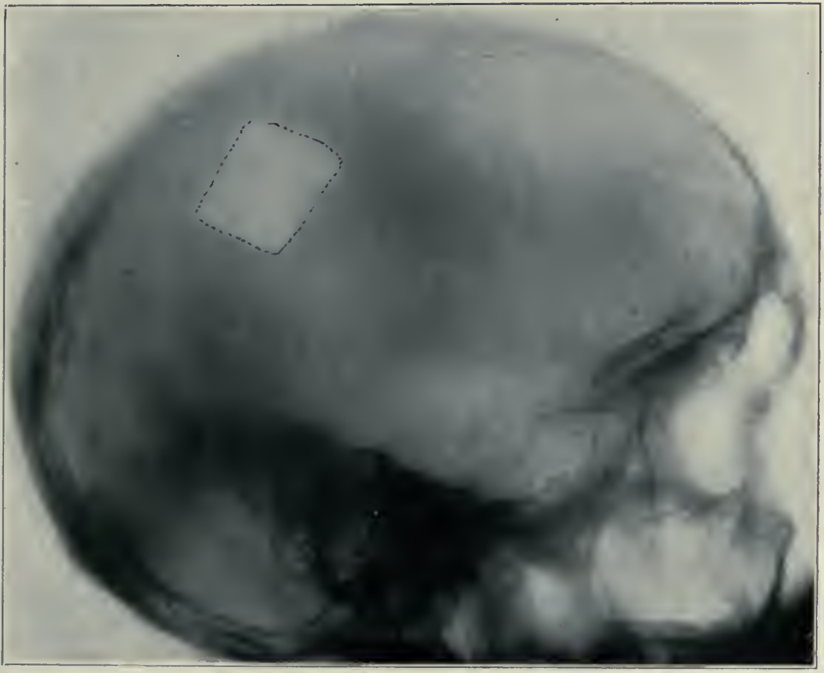

Fig. 7.—Skiagraph of a rectangular defect, in the parieto-occipital region, resulting from removal en bloc of area of skull in the débridement.

Aphasics constituted another group that received special attention. Trained teachers gave the members of this group daily individual instruction and exercise in conversation, reading and writing adapted to the needs of the patient and the character of his language disturbance. Although we have seen no evidence of the development of new language centers on the normal side of the brain, improvement has been marked in every patient of this group, the aphasic symptoms of some of whom had previously remained stationary for several months.

SURGICAL CONSIDERATIONS

To the neurologist the most absorbing features of gunshot wounds of the head are the residual disturbances of function, either local or general. The opportunity for an intensive study of the evidences of perverted function in the individual lobes has been taken advantage of by one of us (S. D. I.), and this aspect of the subject has already been reviewed.

The neurosurgical interest in gunshot wounds of the head attaches to those cases only in which an operation is indicated for the relief of one or the other of the following complications: residual abscess, retained foreign bodies, hemorrhage, epilepsy and cranial defects. These complications will be reviewed in the light of our experience at General Hospital No. 11 with 200 head injuries.

1. *Residual Abscess.*—After ten months of observation we have been able to discover but one case of residual abscess, or 0.5 per cent. A patient who died four days after admission, having been operated on on the transport, is excluded from this category. The necropsy revealed an ill-drained abscess in the occipital lobe, a diffuse basilar meningitis and a large collection of pus in the posterior fossa. That the patient should have survived the journey is nothing short of amazing.

The history of the case (Fig. 1) with the residual abscess was briefly as follows:

History.—The patient was admitted to U. S. Army General Hospital No. 11, Nov. 10, 1918, as an ambulatory case. No record accompanied the patient, and he could give no history of his disability. He had received a gunshot wound of the right frontal region near the midline, date unknown. There was a circular cranial defect about 2 by 2.5 cm. without hernia; the roentgen-ray examination showed several bone fragments near the defect and a foreign body 0.5 by 4 cm. in the parietal cortex above and behind the left ear. No definite motor or sensory or other focal symptoms were present. He appeared to understand what he heard, but refused to speak at first. About two weeks after admission he spoke more freely, but was slightly negativistic.

Treatment and Course.—The sudden development of a hernia at the site of the frontal defect was the indication for an exploratory operation. The site of the hernia in the frontal region was explored for abscess with negative results. The patient subsequently died, and the necropsy revealed a chronic meningitis and an abscess cavity along the tract of the missile filled with inspissated pus (Fig. 2). The contents of the cavity were such that any attempt at drainage would have been ineffective. The symptoms, which were complex, and the cause of the death were attributed to a chronic meningitis and secondary hydrocephalus rather than to the abscess.

Report of Necropsy.—Dura: There was an oval opening just to the right of the midline in the frontal portion, through which the brain herniated. Over the inner surface of the anterior half of the dura, covering the left hemisphere, there was evidence of an internal hemorrhagic pachymeningitis.

Hernia: Beginning at a point 3.5 cm. above the base of the brain, anteriorly and just to the right of the midline, there was an irregularly rounded mass, measuring 3 by 3.5 cm. and projecting 1.2 cm. from the frontal lobe.

Ventricles: The right lateral ventricle was greatly dilated, measuring 11.5 by 4 by 6.4 cm. in its greatest dimensions. At one point the left wall of the anterior horn projected 1.4 cm. to the left of the midline. The ependyma lining this cavity showed irregular areas of grayish white thickening. The choroid plexus of the posterior horn was thickened and firmly attached to the right lateral wall, drawing it inward.

The left lateral ventricle was irregularly dilated, measuring 9.2 cm. in its greatest depth with varying length and width. In the superior frontal region, at one point, the cavity approached to within 0.5 cm. of the surface.

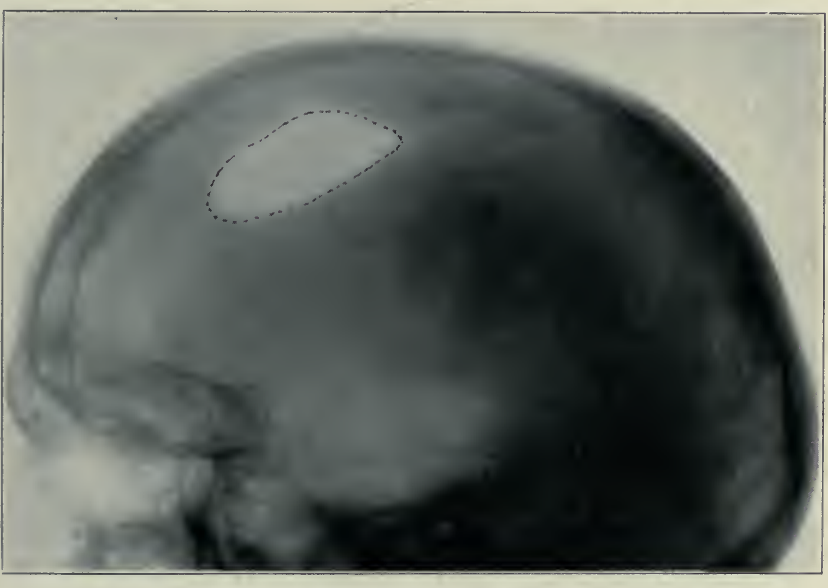

Fig. 8.—Skiagraph of a characteristic oval defect in the frontal region.

Abscess Cavity: Beginning just to the left of the falx, between the frontal lobes and extending outward and backward to the surface in the anterior central gyrus, was a pale orange-yellow granular zone measuring 6 cm. in length and from 1.2 to 2.5 cm. in width, and 3.9 cm. in depth. The central portion of this zone was occupied by a cavity filled with gelatinous and gray amorphous material.

Comparing our figures with those of other countries, we find that, whereas our percentage of residual abscess was only 0.5 per cent., Sargeant and Holmes reported thirty-seven in a series of 1,567 cases, or 1.3 per cent., nearly three times as many. The difference between the two series may be due to the fact that a much larger proportion of cases was evacuated to England after a shorter interval, sometimes ten days or two weeks, whereas with the A. E. F. the interval was longer, and in this interval some cases of residual abscess may have been operated on before transport. I have excluded from consideration fifteen cases of superficial abscesses which were complications of surface infection and osteomyelitis.

Retained Foreign Bodies.—Retained foreign bodies, strictly intracerebral, were observed in 10 per cent. of our series. The fragments, either bone or missile, were of various sizes, single or multiple, superficial or deep (Figs. 3, 4, 5 and 6). In no instance, save two, were they the cause of symptoms. One of these cases has already been described as a case of residual abscess; the other was associaated with a jacksonian epilepsy with the following history:

History.—The patient, who was struck by a high explosive shell in the frontal region just above the eyebrow, received first aid at once. He was operated on at the field hospital; fragments of bone were removed and the wound closed with tube drainage. Sept. 27, 1918, the wounds had healed.

He was operated on June 9, 1919, at General Hospital No. 11. Craniotomy was performed for removal of a foreign body which was localized by measurements and identified by its relation to the bifurcation of the anterior middle meningeal artery, grooves of which could be seen in the skiagraph. The foreign body was located with a needle and extracted with a magnet. It appeared to be covered with soil. The sinus from which it was extracted was disinfected with dichloramin-T and the wound closed. Just before leaving the operating table the patient had a short convulsive seizure involving left face, left arm and left leg.

The foreign body was readily localized and its close proximity to the precentral convolution justified us in the belief that it might have been the exciting factor in the epileptic seizures. At all events this possible relationship, the fact that there had been but two seizures, that the foreign body could be removed without risk to life or harmful damage to the brain, justified its removal.

Our percentage of retained foreign bodies, 10 per cent., is almost identical with that recorded by Sargeant and Holmes; 164 in 1,567 cases, or 11 per cent. That the percentage is so low speaks for the thoroughness and the success with which the original operations were performed at the field and evacuation hospitals. The percentage of fatalities, as given in the British table, was, however, higher than ours. Of their 164 cases, twelve, or 7 per cent., died, whether with or without operation is not stated; of our twenty-three cases, one, or 4 per cent., died.

The indications for operation in the presence of foreign bodies are clearly defined and may be summed up as follows: Those causing encephalitis or epileptic seizures should be extracted and those apparently latent should be left alone. At least these are the principles which have governed us in our selection of cases for operation. I have purposely excluded from our statistics of foreign bodies minute sequestrums or fragments which have been found in sinuses or infected wounds leading to or in the cortex of the brain. There were, all told, seventeen of these, revealed by roentgen ray, and removed merely as a phase of the wound treatment.

Hemorrhage.—In the report of Sargent or Holmes, of the forty-six out of 1,239 cases dying after evacuation to England, eleven were attributed to intracranial hemorrhage. This complication has not been observed in our series.

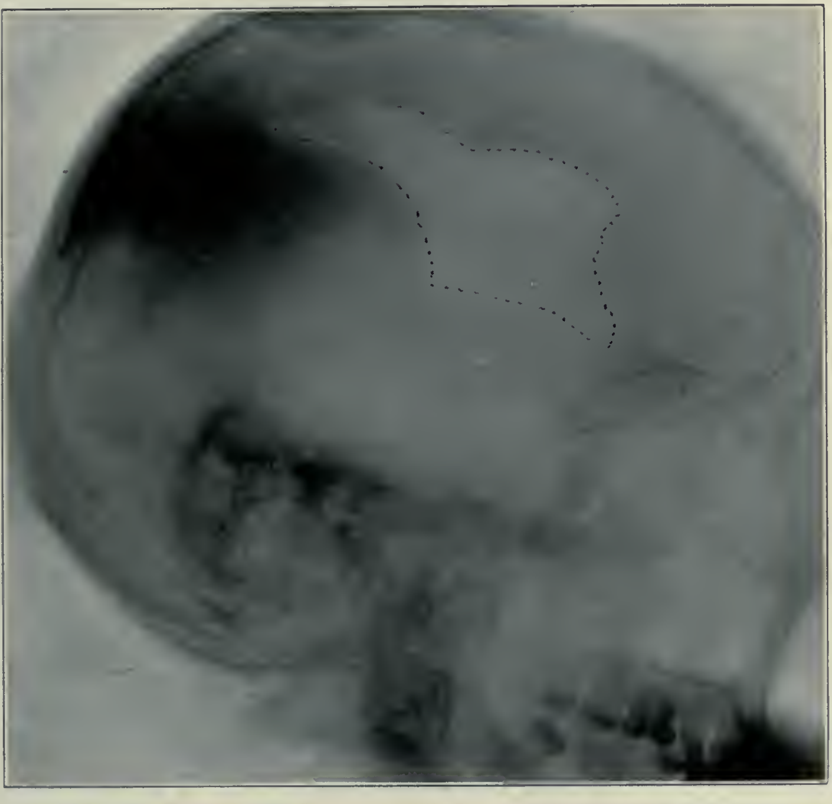

Fig. 9.—Skiagraph of an irregular defect in the parietal region.

Epilepsy.—Whether epilepsy will be admitted as a surgical complication we leave to the reader's decision. There have been so many ill-advised operations for epilepsy on the part of surgeons, that we submit the question with due apologies for their shortcomings, and yet the frequency of epilepsy is such and the resulting disability so great if unrelieved, that the subject cannot be lightly passed over. This complication from its general neurological aspect has already been dealt with, but I will quote a few statistics merely to emphasize its importance.

At the Third Interallied Congress held in Val-de-Grace, Nov. 5, 1917, Tuffier and Guillain reported 676 cases, or 10.14 per cent., in 4,262 cases and in the publication of the Medical Research Committee of Great Britain, Adie and Wagstaffe reported thirty-seven out of 610 cases, or 6 per cent. In our 200 cases there have been twenty-five, or 12.5 per cent. These percentages correspond very closely, but in literature generally one finds an extraordinary variation in the quotations. For example, Villande[1] makes the unbelievable statement that of 450 cranial wounds, in 303, or 70 per cent., secondary operations were performed for which the sole indication was the development of epileptic seizures. Our policy in the matter of operative interference has been briefly this: We have not recommended operation in any case as a preventive measure; we disapprove operation, except in those cases in which the presence of a foreign body or a cranial defect and the resulting dural adhesions might be regarded as responsible for the seizures. Under these restrictions, we have operated on four cases.

Case 1.—The patient, who was admitted on a litter Sept. 25, 1918, was wounded July 18, 1918. He had an infected gunshot wound of the left parietal region with a defect 4 by 5 cm.; hernia cerebri was present. He had a convulsion after admission and curettage of wound Oct. 19, 1918, with a second curettage April 10, 1919. The wound finally healed about May 1, 1919. He has had no convulsion since January and only one after operation.

Case 2.—The patient, who was admitted Dec. 21, 1918, received July 13, 1918, on the Italian front, a gunshot wound in the midfrontal region with a small defect 1 by 1 cm. The wound had healed. The roentgen ray showed a foreign body in the right precentral area about 1 cm. from the dura. The patient showed no paralysis. While on furlough in April he had a general convulsion coming on during sleep, and in May had local jerking of the left arm without loss of consciousness. June 9, 1919, the foreign body was removed. He had three convulsions on the day of the operation but has had none since.

Case 3.—The patient, who was admitted Dec. 12, 1918, received July 4, 1918, a gunshot wound of the right parietal region with a defect 4 by 7 cm. He gave a history of convulsions before admission and had two convulsions December 20. He developed status epilepticus Jan. 20, 1919. Cranioplasty was performed Jan. 21, 1919. He has had no convulsion since.

Case 4.—The patient was admitted Aug. 30, 1918, having received a gunshot wound of the left parietal region and lost his left eye on May 13, 1918. The parietal defect was 4 by 4 cm. He had convulsions soon after admission. Cranioplasty and cartilage graft were performed Oct. 24, 1918. He had one focal convulsion after operation, but has had none since.

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[1] Villandre, C.: Healing of Skull Wounds, Arch. de med. et pharm. mil. **68**:546 (Oct.) 1917.

Late Cerebral Hernia.—It was a source of surprise that we received no cases of cerebral hernia, despite the fact that there were twenty-six cases of open wounds and 153 of cranial defects. Again, to quote statistics, Tuffier and Guillain report 54 in 6,664 cases of gunshot wounds, 0.81 per cent.; in only three of 656, 0.4 per cent., cases evacuated to England, was the hernia larger than a golf ball (Adie and Wagstaffe); in ninety of 1,567 cases, 5 per cent., evacuated to the hospitals of Great Britain, were there large cerebral hernias (Sargeant and Holmes). Needless to say, we would not have included late cerebral hernia among the indications for surgical intervention.

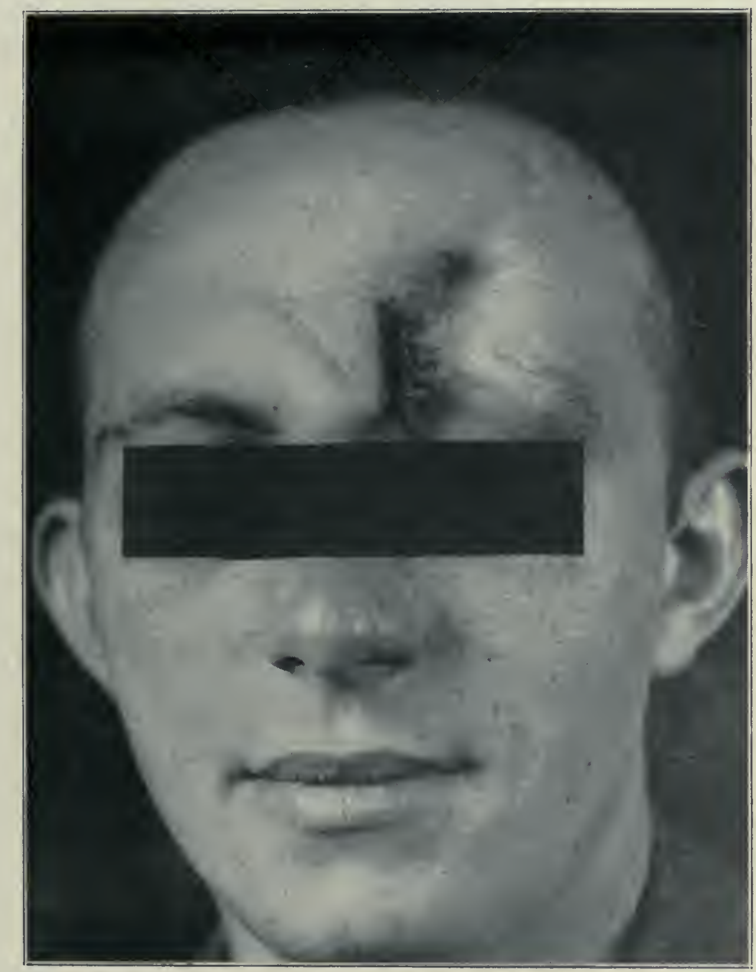

Fig. 10.—Characteristic defect in the frontal region.

Cranial Defects.—With few exceptions—one residual abscess, one intracerebral foreign body and four epilepsies—we have been confronted with no condition justifying resort to operation other than the presence of cranial defects and of these there were, all told, 153 out of 200 cases, or 70 per cent. Of the number, twenty-six were from 1 to 4 cm. in diameter, fifty-one were from 4 to 16 cm. in diameter, thirty-seven were 16 cm. and over in diameter.

The shapes of the defects were round, irregular or rectangular. In all cases the defect followed the deliberate removal of bone as a part of the preliminary débridement; in some instances, fragmentarily; in others, evidently en bloc (Figs. 7, 8 and 9). There was a noticeable difference in the physical condition of the tissues of the integument and underlying it. In some instances there was marked pulsation; in others, not; in some, when the patient stooped, the underlying brain would protrude beyond the level of the scalp; in some the integument contained dense connective tissue which served as adequate protection to the brain; in others the integument consisted solely of a thin reddish cicatrix which often for lack of nutrition exhibited a superficial ulceration. We assumed that in the latter instance there was no dura mater at the site of the defect, that originally there had been a hernia cerebri and, in the process of wound repair, the brain was first covered with a thin layer of granulation tissue, which ultimately cicatrized.

There has been much discussion as to the propriety of repairing cranial defects, even though the operation is, in proper hands, unattended with risk and uniformly successful. First, we will enumerate the conditions under which the operation is not recommended:

1. For the repair of small defects.

2. For the repair of defects in which the brain is adequataely protected by a dense cicatrization and there is no protrusion when the patient stoops.

3. For the treatment of cerebral hernia.

4. For the relief of epilepsy except under the conditions previously noted.

5. When there are any grounds of suspecting an abscess or when there are foreign bodies which may require removal.

The cranial defects should not be repaired, according to Guillain, if the cerebrospinal fluid is not absolutely normal as to tension, chlorid and albumin contents; if there is a pupillary stasis or a choked disk; if there have been partial or general epileptic phenomena; if there are signs of serious meningeal symptoms.

Under what circumstances then is an operation indicated? These may be briefly summarized as follows:

1. For cosmetic reasons, especially when the defect is in the frontal region below the hair line. There are many of these.

2. In certain selected cases of epilepsy.

3. In the presence of a large defect where the brain is exposed to trauma.

4. When the patient is apprehensive, because of the defect and he dreads the possibility of a direct blow on the uncovered cortex.

5. In a few cases where the symptoms are wholly subjective, an operation is advisable in the hope that, combined with the influence of suggestion, the neurosis will be relieved.

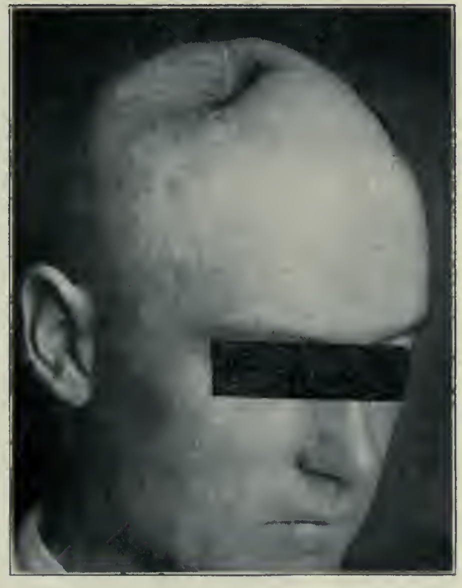

Fig. 11.—Characteristic defect in the parietal region.

Under these indications we have operated on fifty-nine out of 153 cranial defects, or nearly 40 per cent.

There are some who not only do not advise the operation under any circumstances, but who believe operation is distinctly harmful. Dide and Claude thought it psychologically obnoxious, to be a factor in the persistence of subjective symptoms and in producing a neurasthenic syndrome. Andre Thomas said that sometimes epilepsy developed after operation, or if present at the time, was aggravated by it.

We have not included in the indications for cranioplasty the possibility of affording relief to a miscellaneous group of symptoms of which patients often complain who have sustained an intracranial injury. I refer particularly to headache, vertigo, tinnitus, drowsiness, lack of initiative, depression, amnesia and occasionally vomiting. It may be said, however, that not infrequently after an operation for the repair of the defect, there is a manifest improvement in the patient's condition. One of the patients said he had not felt so well since the day he was injured. The headache is less constant, the patient, hitherto depressed, is brighter and more optimistic, he complains less of vertigo, especially on stooping, changing position or taking exercise. I venture on no explanation for this temperamental change, but merely record it as a phenomenon which I had observed previously in the intracranial injuries of industrial accidents and more recently in the gunshot wounds of the head.

Cranioplasty.—Since the outbreak of the war there have been numerous contributions to the subject of cranioplasty. This is quite natural since the opportunity for performing this operation has increased in untold numbers. Many an author writes as though the surgical repair of defects had not been practiced before the war. As a matter of fact, the technic of the operation had been elaborated in every detail and no revision was necessary in its application to the defects due to gunshot wounds. It has been my practice for many years to repair cranial defects with a bone transplant, composed of the pericranium and a thin shell from the outer table of the skull. The operation is devoid of risk, easy of performance and uniformly successful. But in the war literature one finds many other methods recommended, notably the use of cartilaginous grafts and skull fragments taken from the dead. French writers spoke so enthusiastically about their results with grafts of costal cartilage that I was tempted to substitute these for the external table graft which I had been in the habit of using. The immediate results were not as satisfactory in that the cartilage graft collapsed in the wound, and left a shallow saucer-like depression, and of the late results there have been reported instances in which the graft was absorbed. After a brief trial of the cartilage graft I soon abandoned it and resumed my original prewar technic.

A COMPARISON OF THE ANTERIOR HORN CELLS IN THE NORMAL SPINAL CORD AND AFTER AMPUTATION *

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PHILADELPHIA

This work arose from the negative results of an examination, by a student in the department of neuropathology, of three spinal cords from cases having had amputation of a limb sometime previous to death. The findings were so little in accord with the idea of postamputation pathology gained from various textbooks, that a further examination of cords was undertaken in the same manner, from subjects not showing signs of cord lesions during life, and from that point of view called normal. Except for this general precaution material was chosen at random.

A variety of findings are recorded in the literature as a result of investigations of the changes in the anterior horn cells following amputation. No attempt has been made to include a complete literary review; the plan is only to give an idea of the varied results arrived at by a number of experienced investigators.

LITERATURE

Campbell[1] says, after considerable work on this subject, "it may be explained that in consequence of such a lesion alterations occur in the spinal cord and these in course of time occasion striking and characteristic appearances. In long-standing cases the predominant change is a homolateral atrophy, represented by a general reduction in volume of white and gray matter alike, and involving those particular segments of the cord which receive and give off the sensory and motor nerves which originally supplied the skin and muscles of the amputated member. Wasting of the gray substance is accompanied by the numerical reduction of its contained nerve cells, both large and small, and while

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* Contribution in a series offered to Professor E. E. Southard in honor of the decennium of the Bullard Professorship of Neuropathology, Harvard Medical School.

[1] Campbell, A. W.: Changes in the Spinal Cord Following Amputation, British M. J., March 14, 1896, p. 663; Post-Amputation Change in the Cerebral Cortex, Histological Studies on Localization of Cerebral Function, p. 47.

all the cell collections in the anterior cornu suffer, one special group may be singled out as being specially prone to atrophy, namely, the posterolateral."

Marinesco,[2] one of the earlier investigators of this question, is one very often quoted. His report includes three cases: one with duration of twenty-one years; one ten years, and the third of unknown duration. He found changes in anterior horn cells; in the first two cases a reduction in number on the side of the amputation, not only in the lateral, but in other groups; in the third case neither the cell count in the posterolateral group, nor of all cells in all groups have sufficient significance to conclude that there is any definite change in the cells.

Edinger[3] reports a case of prenatal amputation of the left lower arm with death at 52 years of age. The left side of the cord corresponding to the four lower cervical and first two dorsal segments showed a distinct atrophy of the anterior horn, which was greatest at the level of the sixth and seventh cervical segments. There was also a distinct decrease in the size and number of the anterior horn cells, especially at the anterolateral and posterolateral angles of the anterior horn. In three additional cases of postamputation, no convincing proof of cell atrophy was found in the anterior horns.

Van Gehuchten[4] examined the lumbosacral cord from a case twelve years after amputation of the lower extremity in the middle portion of the thigh. The anterior horn cells representing the foot muscles were normal, while the cells representing the muscles below the knee had disappeared, in spite of the fact that the axons of both groups of cells had been divided.

Déjerine and Mayer[5] studied eight cases of amputation varying from four to forty years following amputation. They found in all cases a definite decrease in the size of the side of the cord corresponding to the amputation, affecting both white and gray matter, both anterior and posterior. In only one case were histologic changes found; this was following amputation of the thigh, of thirty years' duration. In the middle of the lumbar enlargement a definite decrease in the number of anterior horn cells appeared, the number being only a third of the normal side, and the anteromesial group was the one most affected.

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[2] Marinesco, G.: Ueber Veränderungen der Nerven und des Rückenmarks nach Amputationen; ein Beitrag zur Nerventrophik, Neurol. Centralbl. **11**:463, 1892.

[3] Edinger, L.: Rückenmark und Gehirn in einem Falle von angeborenem Mangel eines Vorderarms, Virchow's Archiv. **89**:46, 1882.

[4] Van Gehuchten: Handbuch der Anatomie des Nervensystems, **1**:139; Bull. de l'Acad. roy. de Belgique, 1898, p. 222; Presse méd. Jan. 4, 1899.

[5] Déjerine and Mayer: Bull. Soc. de Biol., 1878.

Elders,[6] in an excellent article, reports the examination of the cord of a person born without the left lower arm. Death occurred at 54 years of age. In the lower half of the left cervical segment the posterolateral angle was rounder and shorter than the right and contained fewer cells.

Dreschfeld[7] examined a case of amputation of the left thigh fifteen years after operation, and found atrophy of the anterior horn and some change in the posterior horn in the lower part of the left lumbar enlargement, with decrease in number and atrophy of the nerve cells of the intermediolateral group.

Kahler and Pick[8] report two cases, one of which was an amputation of the lower third of the left thigh, eighteen years after operation. The anterior horn cells were decreased in the anterior group. The second case was an amputation of the left forearm in the lower third. He reports partial atrophy and decrease in the number of cells in the lateral group.

Dickinson[9] reports findings on a case of amputation of the right leg fifteen years before death, in which there was a decrease in the number and size of the nerve cells of the corresponding side.

Genzmer[10] found decrease in size of anterior horn and in the number of anterior horn cells on the amputated side in a case of amputation of the lower third of the right thigh 30 years before death.

Hayem[11] examined the cord in the case of exarticulation of the wrist, five years after operation, and found general atrophy of the anterior horn with atrophy of nerve cells, most marked at the level of the eighth cervical and first dorsal nerves.

Friedreich's[12] case of amputation of the left forearm twelve years before death showed no changes in the cord; the anterior horn cells were intact.

ANIMAL EXPERIMENT

Experimental work also has been done on various laboratory animals, both by section of nerve trunks and by amputation of extremities.

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[6] Elders: Monatsch. f. Psychiat. u. Neurol., 1910.

[7] Dreschfeld: On the Changes in the Spinal Cord After Amputation of Limbs, J. Anat. & Physiol. **14**:424, 1879.

[8] Kahler and Pick: Weitere Beiträge zur Pathologie und Pathologischem Anatomie der Central Nervensystems, Arch. f. Psychiat. **10**:360.

[9] Dickinson: On the Changes Which Occur in the Spinal Cord After Amputation of a Limb, Compared with the Changes Found in Progressive Muscular Atrophy, Trans. of Path. Soc. of London, **24**: 1873.

[10] Genzmer: Veränderungen im Rückenmark eines Amputierten, Virchow's Archiv. **66**:265, 1876.

[11] Hayem: Arch. de Physiol. Norm. et Path., T. V., 1873, p. 504.

[12] Friedreich: Ueber Progressive Muskelatrophie, Berlin, 1873.

Van Gehuchten[4] divided the sciatic nerve and later found only the cells of the posterolateral group affected.

Homén's[13] work on animals showed a slight atrophy and decrease in the number of cells of the anterior horn. The individual cells were found in general somewhat smaller than the corresponding ones on the opposite side. No changes were found in the brain.

The experimental work of Warrington[14] is a more recent and detailed investigation of the changes of the anterior horn cells after section of the spinal nerve roots. In some instances changes were found both in anterior and posterior horn cells corresponding to the side operated on. He reports finding changes not only on the corresponding side, but also on the opposite side. "After section of several posterior roots from the VIth to the IXth post thoracic inclusive, a considerable percentage of obviously altered cells are found. Their distribution in the case of the cat is practically limited to the VIIth and VIIIth segments, and especially to the postero-lateral group of cells in those segments. In the monkey the upper part of the VIIth segment is picked out. The effect is to a very slight extent a crossed one and presents the remarkable feature that more affected cells were found in the VIth segment of the crossed side than on the side of the lesion." Further, "the view I wish to maintain is that the changes are the result of the withdrawal of the afferent impulses which normally impinge on cornual cells. Histological evidence shows that the postero-external group of anterior horn cells is most richly innervated by the collaterals from the posterior roots." He thus explains the degeneration of this group of cells, and adds further with emphasis that the changes occurring after section of the posterior roots are much more intense and that the ultimate destruction of these cells is more likely to occur than after the division of the anterior roots alone. After section of both anterior and posterior roots all the larger cells on the side of the lesion showed changes. The greater number were only slightly affected compared to the extent seen after section of an anterior nerve root alone; others, however, showed a much more marked chromatolysis, the changes resembling those found after section of the afferent roots. He finally concludes that there is a tendency for the altered cells to return to their normal structure regardless of the regeneration of their axons, in conformity with the opinions of Van Gehuchten and Nissl.[15]

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13. Homén: Veränderungen des Nervensystems nach Amputationen in Ziegler: Beiträge zur Pathologischen Anatomie, 1890, p. 304.

14. Warrington: Observations on the Structural Alterations Observed in Nerve Cells, J. Physiol. **23**: 1898.

15. Nissl, F.: München. med. Wchnschr., 1898, p. 6.

FROM TEXTBOOKS

Concerning the change in the anterior horn cells following division of their fibers Oppenheim[16] says: "The Nissl stain shows destruction of the granules and an excentric position of the nucleus. If a restitution in the periphery takes place, regeneration results. If, however, there is no peripheral regeneration, further change in the form of atrophy takes place in the cells."

Van Gehuchten: The section of the axon leads to a rapid swelling of the cell protoplasm, and consequent displacement of the nucleus. He believes that in a small number of cases the swelling of the cell body takes place so rapidly and the force which pushes the nucleus to one side is so powerful that it is driven completely out of the cell body. These cells are the only ones, he concludes, which completely atrophy, while all the others slowly return to normal.

Déjerine and Thomas:[17] For localization studies, division of nerves has been made. In man, the cords of persons having had amputations have been studied with the same end in view, but the application of this method has not always been free from reproach.

Schäfer: If the degeneration has been caused by section of the axon, the reparative process is very slow, so that it may be three or four months before it is completed. At the end of this time the nerve-cell bodies have resumed their original appearance even though reparation of the cut nerve may be incomplete.

Barker:[18] The motor fibers of the central stump gradually diminish in number; in some instances they appear to vanish almost totally, and a large number of the motor cells of the ventral horns dwindle in size and after a time actually may be lost.

MATERIAL EXAMINED

The tissue was prepared in serial paraffin sections, 6 microns thick, and stained by the cresylechtviolet cell-stain method.

Material from three cases of amputation was examined as a starting point of this work, and mainly from the point of view of loss of anterior horn cells. Sections were examined from the segments of the cord corresponding to the part amputated, and the cells of both anterior horns were counted.

Case 1.—Man, aged 79, sixty years after amputation of left arm. The anterior horn cells showed no chromatolysis, but many of the cells were well filled with lipochrome. This is not unusual in view of the age of the man. One hundred and thirty-three serial sections were counted. The variation in individual sections was considerable, but the final average of all counts was, right 17, left 19, or an average of two cells more on the amputated than on the opposite side.

In the brain the Betz cells and other large elements contained much lipochrome, but showed no reaction to the amputation.

Case 2.—Man, aged 65 years, recent amputation of lower third of the left leg. The anterior horn cells were very little altered. There was no chromatolysis, and very little lipochrome. Forty-one serial sections were counted. Variation in individual sections was as much as 62:38 between the two sides. The average of the total number counted was, right 52, left 55, or an average of three more cells on the amputated side. As the operation in this case was performed only the day before death (following gangrene of the foot), no permanent change in nerve cells would be expected. No axonal change had taken place.

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[16] Oppenheim, H.: Lehrbuch der Nervenkrankheiten, **1**:148.

[17] Déjerine and Thomas: La Moelle Epiniere, 165.

[18] Barker, L. F.: The Nervous System, New York, p. 230.

Case 3.—Female, aged 73, old amputation of right thigh. The anterior horn cells contained a great deal of lipochrome, but also some normal Nissl granules. A total of 41 counts was made on serial sections. The variation in individual sections was as great as 13:32. The total average was, right, 21.8; left, 23.9, or an average of two more cells on the nonamputated than on the amputated side.

In addition to these counts others were made on material from a variety of cases, with the single precaution that they show no clinical signs of cord involvement. Both sexes were included, and the ages ranged from 11 months to 71 years.

One case considered particularly suitable for use as a normal control was that of a young male adult, 37 years of age. Here the average of 67 counts in the cervical region was 50 on one side and 54 on the other; of 53 counts in the dorsal region, 12.0 on one side and 12.9 on the other; in the lumbar region the average of 62 counts was 49.5 on one side and 52.0 on the other. This variation is seen to be greater than in the cases of amputation. In Marinesco's[2] work the greatest number of sections of one level counted is sixteen, and the variation in the sum total is 245:223. In the instances where a smaller number of sections were counted, the difference between the two sides was even greater. In the case of the normal control just described, in one group of 21 counts, the total was 938 on one side and 1,181 on the other; but in the final sum of all counts, 67 in all, the number was 2,603 on one side and 2,684 on the other.

In the accompanying table the detail of the entire number of cases counted may be seen. No attempt was made to identify the exact segment of the cord from which the blocks were taken, as much of the material was already embedded without regard to this detail; consequently, there is a great difference in the count at the varying levels. It may be noted also that numbers are much larger up to and including Case 26 than in the subsequent ones. This is due to the fact that in the earlier cases all cells were counted which represented the full contour of a cell including any of its processes, exclusive of whether it contained a nucleus or not. Later, only those cells were counted which contained a stained nucleus. But since the comparison is that of the two sides of the cord, and not between different specimens, the relation is not altered.

TABLE 1.—Counts of Anterior Horn Cells in Serial Sections of Spinal Cord

Case	Diagnosis	Sex	Age	Following Amputation						
				Cervical		Dorsal		Lumbar		Sections Counted
				1	2	1	2	1	2	
1	Amputation, left arm......	M	79	19.3	17.4	....	....	....	....	133
2	Amputation, left leg........	M	65	....	....	....	....	55	52	41
3	Amputation, right leg......	F	73	....	....	....	....	23.9	21.8	41
				Without Amputation						
4	Congenital syphilis.........	F	13	15	16	6	5.7	16.–	17.8	C.15–D.17–L.18
5	Not insane..................	M	6	14.–	18.6	4.9	5.–	18.–*	15.–	C.15–D.15–L.16
6	Organic dementia..........	M	42	23.–	31.8	5.5*	6.–	37.5	28.–	C.16–D.14–L.15
7	Mongolian idiot............	F	6	11.8	12.–	11.6	14.–	28.8*	27.–	C.15–D.26–L.26
8	Idiot........................	M	17	43.–	52.7	10.7	10.8	29.4	31.–	C.18–D.22–L.19
9	Imbecile....................	F	46	36.–	32.–	6.–	6.–	34.–	31.–	C.18–D.17–L.15
10	Alcoholic....................	F	38	30.7*	28.–	6.–*	6.–	33.–*	37.–	C.16–D.17–L.15
11	Auto-intoxication..........	F	42	29.–*	34.–	7.–*	6.–	33.7*	35.–	C.16–D.34–L.18
12	Alcoholic delirium..........	F	32	27.6*	41.6	6.8*	7.–	27.–	28.6	C.15–D.33–L.18
13	Special control..............	M	37	50.4	54.3	12.–	12.9	49.5	52.4	C.67–D.53–L.62
14	Presenile depression........	F	68	23.5*	22.5	4.–*	5.–	19.9*	17.9	C.15–D.15–L.15
15	Unclassified.................	M	71	19.4	19.6	2.8	3.8	19.–	17.8	C.15–D.15–L.15
16	Delirium.....................	F	..	14.9	13.4	2.6	2.5	17.8	19.2	C.15–D.36–L.15
17	Not insane..................	F	51	....	....	8.–	8.6	22.7	21.8	D.34–L.14
18	Dementia praecox..........	F	40	21.–*	25.–	5.9*	5.4	14.–*	11.–	C.21–D.18–L.22
19	Imbecile.....................	M	34	34.3*	21.3	11.3*	6.3	24.9*	23.4	C.20–D.25–L.20
20	General paresis.............	M	39	20.4*	20.2	3.–*	3.1	8.2*	8.5	C.20–D.20–L.20
21	Unclassified.................	M	21	....	....	4.–	4.–	14.–*	13.5	D.41–L.25
22	Chronic dementia...........	F	40	8.9*	8.1	1.8*	3.–	11.–*	9.1	C.31–D.21–L.33
23	Puerperal mania............	F	24	6.6*	7.8	2.1*	2.–	7.4*	8.7	C.21–D.22–L.20
24	Uremic intoxication........	F	28	4.–*	5.6	1.3*	1.9	7.7*	6.6	C.23–D.32–L.21
25	Dementia praecox..........	F	35	6.5*	6.9	1.2*	1.2	10.3*	10.4	C.20–D.22–L.20
26	Imbecile.....................	M	36	5.5*	3.4	2.1*	2.2	....	....	C.16–D.17
27	(Infant).....................	..	11 mo	6.9*	6.2	3.–*	3.8	13.–*	12.6	C.23–D.22–L.22
28	Unclassified.................	F	3 yr. 10 mo	....	....	3.–*	2.7	17.–*	19.–	D.24–L.24

* Left side of cord.

These results contribute something also to the question of whether the number of cells is greater on one side or the other from the standpoint of right and left handedness. It will be seen that there is no constant relation throughout the list, the number being greater as an average on one side in some instances, and less on the same side in another, with very slight variation in any case, and no more in the cervical than in the lumbar region. By means of a low power lens, it may be seen in progressing from one section to another that one horn contains the greater number of cells, first on one side and then on the other, and the same variation is seen in the individual cell groups.

It may be noted here that Bruce[19] found only the fifth cord specimen examined suitable for use in preparing his Atlas of the Spinal Cord.

SUMMARY

Cell counts were made on the anterior horns of the spinal cord in twenty-eight cases. Three of these were cases following amputation of an extremity. The twenty-five additional cases were said not to have had signs of a cord lesion.

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[19] Bruce, A.: Atlas of the Spinal Cord, 1901, p. 3.

Variations in the counts between the two sides in the amputated cases was not more than two or three cells in the final average. In two of these the greater number was on the side corresponding to the amputation.

Variations in the counts between the two sides in the cases without amputation was at times greater than with amputation.

In the sections where identification was made of the right and left sides, there appears no uniform difference between the two corresponding counts.

CONCLUSIONS

The literature contains a variety of opinions concerning the permanent changes in the anterior horn cells of the spinal cord following amputation.

A fairly large number of counts of anterior horn cells of the spinal cord from cases without amputation show as great a variation in the number of cells as in cases with amputation.

In the material examined there seems to be no constant variation between the number of anterior horn cells in the right and left sides of the spinal cord.

NONCONCOMITANCE OF SPINAL FLUID TESTS *

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TESTS APPLIED TO SPINAL FLUID IN DIAGNOSIS OF DISEASE OF CENTRAL NERVOUS SYSTEM

There are five laboratory tests commonly applied to the spinal fluid in the diagnosis of disease of the central nervous system, namely: (1) Wassermann reaction; (2) test for globulin; (3) test for increased albumin; (4) cell count (increase called pleocytosis); (5) Lange's colloidal gold test. A positive Wassermann reaction in the spinal fluid is practically specific for neurosyphilis. A positive result in the other tests, in a general way, proves a pathologic reaction of an inflammatory nature in the central nervous system. At any rate, where an inflammatory condition of the nervous system exists tests 2, 3, 4, and 5 are usually positive. In other words, in meningitis, encephalitis, tumor with meningitis sympathica, vascular insults with secondary inflammatory reaction, traumatic injury, multiple sclerosis, these four tests are generally positive. In paretic or tabetic neurosyphilis all five are usually positive.

The subject of this paper is the consideration of the relation of these tests one to the other, the identity or nonidentity of one to the other and their independent appearance and disappearance in disease conditions. There are three methods of study applicable to this problem: (*a*) chemical and biologic analysis of the substances, (*b*) review of the spinal fluids obtained from a large series of cases with attention to anomalous or unusual relations or nonrelations of findings, (*c*) frequent examination of fluids from cases of neurosyphilis undergoing treatment and in which the spinal fluid findings are influenced in the process of improvement.

WASSERMANN TEST

The Wassermann reaction in the spinal fluid is a biologic reaction pathognomonic of syphilis of the nervous system. As a rule when present it indicates an inflammatory change in the nervous system, but occasionally it is positive when the involvement is chiefly vascular, and not inflammatory. The so-called Wassermann bodies, whatever that substance is which produces deviation of complement, is an unknown element. There is some evidence to show that it is related to the albuminous content of the blood serum and by analogy, therefore, to the albuminous content of the spinal fluid. Thus Bruck has shown that an albumin reaction occurs in many cases giving a positive Wassermann reaction which is different from that obtained in Wassermann negative cases. Weston showed that the Wassermann producing substance does not pass through a dialyzing thimble which is impermeable to albumin. In some unpublished work at our laboratory we have shown that the addition of pure globulin to a spinal fluid giving a negative Wassermann reaction, will cause the reaction to become positive. However, these bits of evidence are far from conclusive that the Wassermann producing body is contained in the albumin and there is evidence, such as that brought forth by McDonagh, that it is a lipoid element with which the Wassermann reaction is associated. The evidence we bring in the body of this paper is against the relation of the Wassermann reaction to globulin or albumin.

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* From the Massachusetts State Psychiatric Institute, Boston.

* A paper presented to Dr. E. E. Southard, in honor of the decennial of the Bullard Professorship of Neuropathology at the Harvard Medical School.

It is of course evident that there is no relation between the Wassermann reaction and pleocytosis. Pleocytosis is evidence of inflammation, the Wassermann reaction of syphilis. Even where the inflammation is of syphilitc origin, the Wassermann reaction may be negative in the presence of a pleocytosis or, what is much more common, the pleocytosis may be absent with a positive Wassermann.

TEST FOR GLOBULIN

Globulin by definition is an albumin which is salted out of solution by half saturation with ammonium sulphate. Globulin does not occur normally in the spinal fluid. When present it is an evidence of pathology, and it is usually considered as proof of an inflammatory type of reaction. It also may be present, however, when there has been seepage of blood into the spinal fluid through a hemorrhage or vessel changes. If globulin is present in the spinal fluid, theoretically it means that there is an increase in the total amount of albumin, as this is an addition of an albuminous substance to the albumin normally present. As albumin which is precipitated by an acid such as trichloracetic, is normally present in the spinal fluid in small amounts, it is increased in amount whenever globulin is present. However, it is not dependent for its increase solely on globulin, but is increased as a result of inflammation. While we have mentioned that increased globulin cannot exist without a theoretic increase in the amount of albumin, albumin can be present in increased amount without the presence of globulin. We will later show that this actually occurs. While again the globulin, albumin and pleocytosis are evidence of inflammation, it does not follow *a priori* that they are dependent one on the other or must be present together. They are quite different elements. The same is true of the Wassermann in relation to globulin and albumin.

COLLOIDAL GOLD TEST

The chemical nature of the substance which produces the colloidal gold reaction is not clearly defined. The colloidal gold reaction is a method of differentiating albumins and was used by Lange in the endeavor to classify the type of globulin occurring in the spinal fluid. Without having succeeded in making this classification, he did discover this practical test. An obvious assumption is that the test is dependent on the albumins or globulins in the spinal fluid. It is probable that the actual type of reaction obtained is dependent to some extent on the balance of globulin and albumin present in the fluid. However, it seems to be a fact that it is not the globulin ordinarily present that causes the precipitation of the gold. Matskiewitsch attempted to identify the substance causing the colloidal gold reaction as peptone. Weston disagrees with this finding. Our clinical experience has shown that the gold reaction may be obtained with a fluid that gives no precipitation with half saturated ammonium sulphate.

Our assumption in this consideration is that these substances may appear independently one of the other, and that while on the whole they represent reaction substances of inflammation, they are in some respects entirely different responses.

SPINAL FLUID FINDINGS

Now if this be so, that these substances may occur independently one of the other (with the one exception that globulin means an increase of albumin) it would follow that a number of combinations can occur, and as a matter of fact do occur. Our evidence is derived from two sources—the routine examination of several thousand fluids and examination of spinal fluids from cases of neurosyphilis undergoing treatment.

From the first group are obtained instances of the independent appearance of the several elements and their appearance in various combinations. From the cases under treatment one finds that one test or another disappears first, and finally only one may be left positive. The different combinations appearing in these two groups are shown in the accompanying table.

Brief examples may be given of the reactions occurring in Group 1. The Wassermann reaction occurs independently of other tests, not infrequently in the endarteritic forms of cerebral syphilis. It occasionally is found as the only positive test in cases of congenital

Results of Spinal Fluid Tests

Spinal Fluid Finding	Condition in Which This Finding Occurred (Untreated Cases)	Findings Occurring in Treated Cases of Neurosyphilis
Pleocytosis	Congenital syphilis, undiagnosed mental disease	
Albumin	Trauma, Korsakoff's psychosis, undiagnosed mental disease	
Wassermann reaction	Vascular neurosyphilis, congenital syphilis	General paresis
Colloidal gold test	Undiagnosed condition	General paresis, tabes, etc.
Pleocytosis and albumin		
Pleocytosis and Wasserman reaction	....	General paresis, cerebrospinal syphilis
Pleocytosis and colloidal gold test	....	General paresis
Globulin and albumin	Cerebrospinal syphilis, trauma, cerebral hemorrhage, brain tumor, chronic alcoholism	General paresis
Albumin and Wassermann reaction		
Wassermann reaction and colloidal gold test	....	General paresis
Albumin and colloidal gold test	Undiagnosed mental disease	Cerebrospinal syphilis
Pleocytosis, globulin and albumin		
Pleocytosis, albumin, Wassermann reaction	Cerebrospinal syphilis	General paresis
Pleocytosis, albumin, colloidal gold test		
Pleocytosis, Wassermann reaction, colloidal gold test	Neurosyphilis	
Globulin, albumin and Wassermann reaction	....	General paresis, cerebrospinal syphilis
Globulin, albumin and colloidal gold test	Multiple sclerosis	Tabes, cerebrospinal syphilis
Albumin, Wassermann reaction and colloidal gold test		
Pleocytosis, globulin, albumin and Wassermann reaction.		
Pleocytosis, albumin, colloidal gold test and Wassermann reaction	....	General paresis
Pleocytosis, globulin and colloidal gold test	Multiple sclerosis, brain tumor, apoplexy	General paresis
Globulin, albumin, Wassermann reaction and colloidal gold test	General paresis, cerebrospinal syphilis	General paresis
Pleocytosis, globulin, albumin, Wassermann reaction and colloidal gold test	General paresis, tabes, cerebrospinal syphilis	General paresis, tabes, etc.

syphilis, tabes and diffuse neurosyphilis. On the other hand, we have seen cases in which it was the only test to be absent. This of course is always true in nonsyphilitic inflammatory conditions giving a positive reaction for the other tests. We have also seen it even in cases of general paresis, tabes and congenital syphilis.

Pleocytosis is the most variable of the abnormal findings. It is not infrequently absent in the chronic inflammatory processes, e. g., in general paresis, tabes, brain tumor and multiple sclerosis, when the other tests are positive. On the other hand, it may be present in the absence of all other tests. For example, a woman suffering from glycosuria and a mild depression showed twelve cells per cubic millimeter; a second puncture showed sixteen cells per cubic millimeter. Her husband was a tabetic. A second example is that of a 20-year-old boy with all the signs of congenital syphilis, with outbreaks of violence, confusion and amnesia. He showed fifty-six cells per cubic millimeter, the other spinal fluid reactions being negative.

An increased amount of albumin as the only abnormal finding is not infrequent. In many instances where one finds this as the only evidence of pathology, one is at a loss to be able to interpret it, and we have seen frequent examples of this sort in dementia praecox and unclassified psychoses. According to Myerson an increased amount of albumin is common in Korsakoff's psychosis. I have been able to confirm this in a very small minority of Korsakoff cases. It also occurs after fractured skull and after fairly recent hemorrhage.

The gold reaction may occasionally be found as the only positive test, though this has been quite rare in our experience. We have found it in a few psychotic cases in which we were unable to explain its significance and in a few cases of syphilis.

It seems unnecessary to go into detail as to the combination of these findings with their clinical significance. It suffices to indicate on the table in what conditions we have found these to occur.

In cases of neurosyphilis receiving intensive antisyphilitic treatment, there usually is a change in the laboratory tests. We find that there is no general rule as to which test is the first to become weakened or to disappear, or as to which will remain positive. As one would expect, they do not all change at identically the same time. Thus in cases of general paresis undergoing treatment the Wassermann reaction is frequently unchanged despite long and intensive arsenic and mercury injections and may remain positive after all other tests have become normal. In another case with the same diagnosis and similar spinal fluid findings before treatment, the Wassermann reaction may be the first test to become negative. The same variation is to be found with the colloidal gold test, globulin and albumin.

In the table (third column) we have indicated the combination of findings that we have seen in treated neurosyphilitic cases. In addition a few examples are given in more detail.

REPORT OF CASES

OBSERVATION 1.—*Wassermann Reaction Remained Positive—All Other Tests Became Negative.*—Woman, 57 years of age, diagnosed as a case of general paresis.

Before treatment: Wassermann reaction +; globulin + + +; albumin + + +; cells 22 per c.mm.; colloidal gold test, general paresis.

After treatment: Wassermann reaction +; globulin 0; albumin negative; cells 0 per c.mm.; colloidal gold test, negative.

OBSERVATION 2.—*Colloidal Gold Test Remained Positive — All Other Tests Became Normal.*—Diagnosis: General paresis.

Before treatment: Wassermann reaction +; globulin + + +; Albumin + + +; cells 37 per c.mm.; colloidal gold test, general paresis.

After treatment: Wassermann reaction —; globulin 0; albumin negative; cells 3 per c.mm.; colloidal gold test, general paresis.

OBSERVATION 3.—*Pleocytosis and Wassermann Reaction Remained Positive; Other Tests Negative.*—The patient was a man of 35 with numerous neurasthenoid complaints, blood and spinal fluid positive to all tests. After two years of treatment spinal fluid findings were: Wassermann reaction +; globulin 0; albumin negative; cells 110 per c.mm.; colloidal gold test negative.

OBSERVATION 4.—*Globulin and Albumin Remained Positive; Other Tests Negative.*—Diagnosis: General paresis.

Before treatment: Wassermann reaction +; globulin + + +; albumin + + +; cells 90 per c.mm.; colloidal gold test 5 5 5 5 5 5 3 0 0 0.

After treatment: Wassermann reaction —; globulin + +; albumin + +; cells 3 per c.mm.; colloidal gold test negative.

OBSERVATION 5.—*Colloidal Gold Test and Wassermann Reaction Remained Positive; Other Tests Became Negative.*—Diagnosis: General paresis.

Before treatment: Wassermann reaction +; globulin + + +; albumin + + +; cells 22 per c.mm.; colloidal gold test, general paresis.

After treatment: Wassermann reaction +; globulin 0; albumin negative; cells 0 per c.mm.; colloidal gold test, general paresis.

OBSERVATION 6.—*Albumin and Colloidal Gold Test Remained Positive; Other Tests Became Negative.*—Diagnosis: Cerebrospinal syphilis.

Before treatment: Wassermann reaction +; globulin + + +; albumin + + +; cells 11 per c.mm.; colloidal gold test 5 4 3 2 2 1 0 0 0 0.

After treatment: Wassermann reaction negative; globulin 0; albumin +; cells 3 per c.mm.; colloidal gold test 0 1 3 4 3 1 0 0 0 0.

OBSERVATION 7.—*Globulin, Albumin and Wassermann Reaction Remained Positive — Pleocytosis and Colloidal Gold Test Became Negative.*—Diagnosis: General paresis.

Before treatment: Wassermann reaction +; globulin + + +; albumin + + +; cells 45 per c.mm.; colloidal gold test 5 5 5 5 5 5 5 2 0 0.

After treatment: Wassermann reaction +; globulin + +; albumin + +; cells 4 per c.mm.; colloidal gold test negative.

Observation 8.—*Globulin, Albumin and Colloidal Gold Test Remained Positive — Wassermann Reaction and Pleocytosis Became Negative.*—Diagnosis: Cerebrospinal syphilis.

Before treatment: Wassermann reaction +; globulin + + +; albumin + + +; cells 56 per c.mm.; colloidal gold test, 0 0 1 3 3 2 1 0 0 0.

After treatment: Wassermann reaction negative, globulin +; albumin +; cells 0 per c.mm.; colloidal gold test 2 2 2 3 3 1 0 0 0 0.

Observation 9. — *Wassermann Reaction Became Negative; Other Tests Remained Positive.*—Diagnosis: Cerebrospinal syphilis.

Before treatment: Wassermann reaction +; globulin + + +; albumin + + + +; cells 41 per c.mm.; colloidal gold test, general paresis.

After treatment: Wassermann reaction —, globulin + +; albumin + +; cells 9 per c.mm.; colloidal gold test, syphilitic.

Observation 10.—*Pleocytosis Reduced to Normal; Other Tests Remained Negative.*—This is a very frequent finding.

Even though not a part of this discussion, we would like to add that the Wassermann reaction in the blood serum varies independently of the spinal fluid findings. Thus it may become negative while all the spinal fluid tests remain unchanged or it may remain positive when all the spinal fluid tests become negative. It is also pertinent to note that the changes in these spinal fluid tests do not always parallel clinical changes in the patients. Thus we have seen illustrations where although the tests became negative the symptoms were in no way improved; and on the other hand, where there was marked symptomatic improvement without any change in the tests.

SUMMARY

Of the five spinal fluid laboratory tests commonly used, only cells and albumin are normal constituents of the fluid, and the excess amounts of these are evidence of pathology. The Wassermann reaction is for practical purposes specific for syphilis of the nervous system. Pleocytosis, globulin, albumin and colloidal gold reaction are generally indicative of an inflammatory reaction of brain, cord or meninges. These tests may be positive owing to infection, mechanical injury, tumor, trauma, vascular insults, multiple sclerosis, etc. As a rule, globulin, albumin increase, pleocytosis and a positive colloidal gold reaction occur together and when the Wassermann reaction is positive, it is usual for the other four to be positive also. Although usually present together and in a general way indicative of the same pathologic condition, each reaction is produced by a distinct chemical element which may be present alone (except that theoretically globulin means an increase in the total amount of albumin, as globulin is a special albumin). Thus one may find the Wasserman reaction positive, all other tests negative, a positive colloidal gold reaction as the only positive finding, only a pleocytosis or merely an albumin increase.

Further, they may occur in various combinations. This is theoretically possible on the basis of difference in chemical constitution, and it is shown in this paper that this actually does occur.

Additional evidence of the independence of each element is offered in the result of treatment of cases of neurosyphilis. Starting with all tests positive, it is shown that they become negative often one at a time and the different combinations are left positive, or only one is left positive.

CONCLUSIONS

1. There is a nonconcomitancy of the inflammatory elements of the spinal fluid commonly tested for in diagnosis of disease of the central nervous system.

2. Any one may be present or absent when the others are present—with the exception that globulin presages an increased amount of albumin.

3. No spinal fluid can be considered negative in which all these tests have not been used.

4. No one element tested for contains the element or fraction that gives another test, except that the total albumin contains the globulin fraction, in part at least.

5. In neurosyphlitic cases receiving treatment these substances disappear at differing rates which vary in different cases, so that no general law can be laid down as to which element is most easily affected by treatment in any particular case, though in general the pleocytosis disappears first.

6. The presence or absence of these products of inflammatory reaction does not always parallel the clinical change in the treated neurosyphilitic patient.

INFLUENZA PSYCHOSES IN SUCCESSIVE EPIDEMICS *

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Psychoses following influenza, as precipitated by the epidemic of 1890-1892 and reprecipitated by the epidemic of 1918-1919, make a particularly interesting study. Among the nearly 200 cases of post-influenzal psychoses studied at the Boston Psychopathic Hospital during the past year, only two such cases were observed. A third case, in which a psychosis had followed the earlier epidemic, was received at the hospital during this period, but without the history of recent influenza. These three cases are reported below.

These cases are interesting because of the question of specificity which Kraepelin[1] raised. He maintained that each acute infectious disease was probably capable of producing mental symptoms of a peculiar sort, a virtual "influenza psychosis," "typhoid psychosis," etc. This view has never been well supported, and as yet we know only that the psychoses following influenza are numerous and of great variety.[2] This might connote a quantitative specificity as contrasted with Kraepelin's qualitative specificity. In the case of many, some writers[3] say nearly all persons, influenza has been followed by states of depression, irritability, irascibility, deconsolateness, etc., which undoubtedly represent *formes frustes* or mild forms of the psychoses appearing in bolder form in less fortunate subjects.

Why some persons should be particularly subject to the neurotoxin of influenza as these cases here would indicate, is as yet unexplained. In the cases cited below there must have been, during the three decades intervening between the two attacks of influenza, both of which were followed by such striking mental reactions, many incidents and agents capable of more or less psychopathologic damage; yet apparently no cause produced a result such as was wrought for the second time by influenza, in the short course of a few days.

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* This is the fourth of a series of papers on influenza and mental diseases from the Boston State Hospital, Psychopathic Department, Series 1919.

[1] Kraepelin, E.: Kraepelin advanced this theory during his early work on the "symptomatic psychoses," but summarizes his views in his Psychiatrie, ed. 8, Leipzig and Vienna, 1912.

[2] Menninger, Karl A.: Psychoses Associated with Influenza, II. Specific Data. An Expository Analysis, Arch. Neurol. & Psychiat. **2**:291 (September) 1919.

[3] Pritchard: Nervous Diseases and Psychoses Following the Grippe, Internat. Clin. XI, ser. 5; Osler: Practice of Medicine, New York.

Another significance of these cases lies in their bearing on the relationship of the infection (toxic) psychoses to schizophrenia (dementia praecox). Postfebrile delirium and schizophrenia were by far the most numerous psychiatric sequelae of influenza.[4] There is, moreover, the notorious[5] similarity in symptomatology of catatonia and hebephrenia to the "postfebrile deliria." On this, in part, is based the thesis, by no means new, that delirium and some forms of schizophrenia may have a similar pathologic basis, and hence etiology. Cases such as those I present, in which an infection (influenza) or some other incident is followed by a psychosis which improves, only to reappear under renewed conditions of deleterious influence (here influenza, except in the third case) suggest the chemical process of reversibility, and specifically the reversibility or partial reversibility of schizophrenia. By extension of the hypothesis of the similar etiology of delirium and schizophrenia, and comparison with the phenomenon just noted, one can conceive that postfebrile deliria may represent, as the writer would propose, a form of reversible schizophrenia.

REPORT OF CASES

CASE 1.—A man, aged 49, with an unfavorable family history, was normal until he had an attack of influenza at 25. He was psychotic for a short time afterward, and had been queer for the past few years. He has had frank psychosis with conduct disorder since he had influenza in 1918.

Family History.—This embraces a psychotic father ("asocial, queer, brooded, suicidal"), an insane cousin, other "high strung" cousins, and a mother who had "a nervous breakdown." He was an only child.

Personal History.—He was not considered queer, asocial, stupid or abnormal either before or after the first attack of influenza. The medical history was negative; his habits were good. He did not marry.

First Attack of Influenza.—"About twenty-five years ago" he is known to have had influenza at the time of "an epidemic." Thereafter he was for a time "affected in his head," at least for a few months. In subsequent years he was very irregular at work, changing jobs often, usually without adequate cause.

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[4] Fell, E. W.: Postinfluenzal Psychoses, J. A. M. A., **72**:1658 (June 7) 1919; Burr, C. W.: Mental Complications of Influenza, Med. Clin. of N. America **2**:709 (November) 1918; Harris, A. F.: Influenza as a Factor in Precipitating Latent Psychoses, Boston M. & S. J. **158**:22 (May 29) 1919; Menninger, Karl A.: Psychoses Associated with Influenza, I. General Data: Statistical Analysis, J. A. M. A., **72**:235 (Jan. 25) 1919.

[5] A point made by many writers, old and new. For example, see the textbooks of Kraepelin, Binswanger, Siemmerling, Paton, Regis; the monographs of Bonhoeffer and of Bleuler; numerous abstract discussions such as those of Hoch, Deny, Dide and others, and finally, the recent clinical observations and reports of Austregesilo, Harris, Fell, the writer and others.

For some years he has been rather paranoid, suspicious and queer, and has begun to do peculiar things, which, however, have been overlooked. Fits of anger and destructiveness, with attitudinization have also been noted. These symptoms have developed gradually during the last seven years, but they did not become serious until after the influenza.

Second Attack of Influenza.—This occurred in October, 1918. Thereafter his psychopathic condition became much worse. He manifested (for the first time) delusions and hallucinations, enfeebled intellectual processes and an emotional tone varying from silly euphoria to causeless agitation. Silliness was manifested in manner, speech and conduct.

Physically, he showed unequal pupils, slurred speech, hypo-active left ankle jerk, and right facial paresis, but otherwise nothing abnormal. All laboratory findings, including serum Wassermann and spinal fluid, were negative.

At first the case was considered, *a priori,* one of neurosyphilis, but laboratory findings disproved this, and the patient was considered merely hypophrenic. (His psychometric rating was 8.7 years, variation total of 17). However, increasing symptoms changed the diagnosis. He became more and more silly; he sat about the ward in fixed positions and without speaking a word for days at a time; he became mute and totally inaccessible. He voiced delusions of being "butchered" and "put in straight jackets." He became steadily worse mentally and was transferred, after six weeks' observation, to the Boston State Hospital.

Diagnosis.—Schizophrenia hebephrenica.

Case 2.—A woman, aged 37, had influenza at 10 followed by psychosis and commitment to an asylum for ten years. She absconded and lived at large, being self-supporting for ten years, when a second attack of influenza (1918) precipitated symptoms requiring commitment.

Family History.—She had an alcoholic father and a sister, now 45, who has recently been committed to an asylum as insane; she is self-accusatory and depressed.

Personal History.—Birth and childhood were negative. She progressed to the third grade, and was considered healthy. She had diphtheria.

First Attack of Influenza.—At the age of 10 (1891) she had influenza. Not many details are known, save that thereafter she was "different"—she was "strange, contrary, ill natured and did not get on well with other children." She claimed that she was discriminated against, that she was abused, that her employers persecuted her and that people were jealous of her.

Finally, she was committed at about 17 years of age. She remained in the state hospitals for some ten years; then she absconded, and was taken in by some family. A friend of her family got in touch with her and heard from her frequently. She lived at large with some fair degree of success, but changed employment very frequently. This continued for ten years.

Second Attack of Influenza.—She was taken to the City Hospital with influenza, Jan. 18, 1919. She recovered and was up and about the ward, but "made irresponsible statements and was excitable." She was transferred to the Boston State Hospital.

She expressed great perturbation over being here, and hastened to assure every one that she was quite free from any mental trouble. She cooperated poorly and was evasive, arrogant, egotistic and irritable. She was seclusive, irascible and unpopular with the patients, and was antagonistic toward everything—physicians, nurses, baths, occupational therapy, etc. She was notably dis-interested, and showed a poverty of ideation and emotion. No delusions or hallucinations were elicited.

Her psychometric rating was 10.3, with a variation total of 15, which is, of course, quite high. She kept saying, "Now I want you to tell me if it is not correct, for I'm perfectly willing to verify it."

Physical examination and laboratory findings, including fluid findings, were entirely negative.

Diagnosis.—Schizophrenia hebephrenica.

Case 3.—A woman, aged 48, supposedly well educated until she had an attack of influenza in 1890, thereafter was gravely demented, but was useful as a domestic. In recent years she has become increasingly queer, and finally totally incompatible. This is a typical case of old schizophrenia. She probably did not have influenza in the recent epidemic.

Family History.—Nothing abnormal was revealed.

Personal History.—She is said to have been "a very well educated woman, and perfectly normal until she had the influenza during the epidemic of 1890."

First Attack of Influenza.—"The influenza struck her brain." She was "just like a child . . . quiet . . . never speaks unless spoken to. Wasted her money and threw away or tore up what she bought. Always talked incoherently." She was employed for the past twenty years by a family from whom this (the only) information was obtained.

Present Episode.—There is no record of her having had influenza during the 1919 epidemic. For a year she had been acting more and more strangely, ceased bathing herself, began to answer her mistress irrelevantly, etc. On the day of admission she "suddenly began to kick the puppy" and frightened her mistress, so that she was brought to the Psychopathic Hospital.

Mental examination showed marked dilapidation of all thought processes; disorientation, delusions of transformation, hallucinations, profound apathy, hypokinesis, and occasional resistiveness and negativism.

Physical examination and laboratory findings were wholly negative.

Diagnosis.—Schizophrenia hebephrenica.

A BRIEF RÉSUMÉ OF NEUROLOGIC AND PSYCHIATRIC OBSERVATIONS IN A HOSPITAL CENTER IN FRANCE

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This résumé includes a statistical report, with brief citations and conclusions drawn therefrom, of cases observed at Hospital Center Mar-sur-Allier, France, during the period from September 18 to Dec. 31, 1918. During that period approximately 25,000 patients were admitted to the Center. They were received as early as forty-eight hours after engagement. At the time of the armistice there were operating in the Center seven hospitals with individual capacity of 2,000 patients, and a convalescent camp with a capacity of 5,000 patients. Each hospital and the convalescent camp had its individual psychiatrist. Each hospital designated a special ward for functional and psychiatric cases. Brain, spinal cord and peripheral nerve injuries were not designated a special ward, but were seen and diagnosed by the respective neurologists.

The total number of neurologic and psychiatric cases observed was 507, classified as follows:

TABLE 1.—Neurologic and Psychiatric Cases

Brain injury	20
Spinal cord injury	17
Cerebral concussions	54
Spinal cord concussions	8
Peripheral nerve injuries	266
Neuroses	103
Psychoses	23
Epilepsy	6
Mental defectives	10

BRAIN INJURIES

Of eighty patients admitted to Base Hospital No. 48 with gunshot wounds of the skull, ten, or 12.5 per cent., showed evidence of injury to the brain substance in these locations:

TABLE 2.—Cases of Brain Injuries

Frontal	3
Parietal (motor, sensory, aphasic symptoms)	3
Parietal (motor symptoms only)	1
Occipital	3

All of the occipital injuries resulted in infection, two developing abscess of the occipital lobe, and one a purulent meningitis at the site of a previous decompression. (One occipital case in Base Hospital No. 14 also developed an abscess). One frontal injury resulted in an abscess. The great frequency of occipital infection was a matter of interest, perhaps explained by the great vascularity of this area.

I was particularly impressed by two objective symptoms which were present in the frontal cases, symptoms which I had previously observed in civil practice. These symptoms were "stuporous states" and "yawning." They were not observed at all in the parietal cases nor was there any yawning in the occipital cases, and stupor appeared only just before death.

REPORT OF CASES

Case 1.—*History.*—One of the occipital cases was in reality one of late abscess and was quite interesting. This soldier was admitted as an ambulatory patient. There was a closed wound over the left occiput and from a pin-point opening in the lower angle of the wound exuded a very slight amount of serous fluid. He was most carefully examined for evidence of hemianopsia, but none was found. For several days he roamed around like any other convalescent wounded patient, with no complaints whatsoever.

Course and Treatment.—Suddenly one day he complained of intense headache. His temperature at 2 p. m. was reported to be 104 F. I saw him about 9 p. m. the same evening, and his temperature was 99.5 F. His headache had disappeared, and he said he felt "all right." On examination he showed complete left homonymous hemianopsia. This was the only objective finding. The next morning his headache had entirely disappeared and he felt quite well. The hemianopsia had entirely disappeared. For ten days thereafter there were absolutely no objective or subjective symptoms. He was repeatedly examined for hemianopsia and none was found. His headaches then suddenly reappeared as did also his hemianopsia. Vomiting became a distressing symptom; his condition progressed from bad to worse. Surgical exploration revealed an abscess of the left occipital lobe. He died seven days later and necropsy revealed the abscess and beginning purulent meningitis. The interesting feature in this case was the behavior of the hemianopsia, which can probably be explained on a vascular basis.

Case 2.—*History.*—A brief notation in regard to the frontal abscess case. This soldier was admitted in a stuporous state, but during the first few hours could be aroused to take nourishment. He "yawned" considerably. All the deep reflexes were normal, the abdominals and plantars were also normal. He died seven hours after admission.

Necropsy.—This revealed fracture of the left frontal bone, the entire left frontal lobe a pulpy abscess mass; petechial hemorrhages; no evidence of infection in the brain stem; a large "spread out" blood clot over the right occipital lobe; slight reddening of the anterior border of the left parietal lobe, but no petechial hemorrhages or evidences of infection within it. There was no communication established between these various lesions. It will be observed that the direction of the force of the trauma was from before downward, inward and backward. Apparently there were three distinct pathologic conditions: First, a fracture of the skull with direct injury to brain tissue followed by infection and abscess; second, general concussion (commotion) causing the petechial hemorrhages; third, the contrecoup phenomenon manifested by the findings in the occipital region.

SPINAL CORD INJURIES

The location of these injuries was about equally distributed between the dorsal, lumbar and sacral regions. I saw no cervical injuries. Where there was complete section of the spinal cord death intervened usually from local infection, or infection involving the genito-urinary tract. Death occurred earlier in mid-dorsal regions than in any other location.

CEREBRAL CONCUSSIONS

On this subject some probably will take issue with me. However, the cases here classified as concussion presented identical symptomatology, namely, occipital pain, mental confusion of varying degrees, mental and physical fatigability, following a definite history of a period of unconsciousness varying from one to twenty-four hours, resulting from explosions. No external injuries were present. Owing to the late period at which these cases were received, lumbar puncture was of little assistance. In a majority of the cases there was an overfilling of the retinal vessels and in some there was clouding of the nasal half of the optic disk and a strong suggestion of weakness of both sixth cranial nerves. Except for hyperactivity of the deep reflexes and fine rhythmic tremors, further neurologic examination was negative. In this class there was no attempt at "conversion," nor could any conscious or subconscious desire or fear be elicited. It was significant that after the cessation of hostilities the number of patients of this class arriving in this Center was considerably diminished.

The value of lumbar puncture at the earliest possible moment in suspected cases cannot be overestimated. In acute confusional cases of any degree, early performed, it will often eliminate a purely functional or psychic condition, and, inasmuch as concussion cases often develop the syndrome of traumatic neurasthenia, the early and routine use of lumbar puncture as a diagnostic aid will often protect these sufferers from being stigmatized "fakers" or seekers after money.

SPINAL CORD CONCUSSION

As to the existence of clinical phenomena following concussion of the spinal cord all are agreed, but as to the actual pathologic tissue condition, there is a difference of opinion. I am inclined to support the theory of necrosis as promulgated by H. Claude and J. Lhermitte. Corroborative evidence of this assumption is furnished by the fact that although lumbar puncture was performed promptly in the majority of our cases, the result of examination of the fluid was negative. The one necropsy which we were able to get revealed no macroscopic abnormalities in the cord. The pronounced clinical disturbance made a hemorrhage, later absorbed, most unlikely.

REPORT OF CASES

Case 1.—Capt. X, thrown several feet by the explosion of a shell, received no external injury. On admission to hospital he suffered from incontinence of urine and feces and a spastic paraplegia with complete anesthesia to the level of the tenth dorsal vertebra. Roentgen ray and lumbar puncture were negative. The incontinence disappeared within twenty-four hours, followed by gradual disappearance of sensory and spastic phenomena.

Case 2.—B., who had received a gunshot wound in the lumbosacral region and was admitted to hospital as an ambulatory patient, presented typical drop-foot gait, and gave a definite history of rectal and vesical incontinence. Examination revealed a double drop-foot, and knee jerks were present and equal. The ankle jerks were not elicited and there was hypothesia over the entire sacral segment on both sides. Lumbar puncture was negative. Roentgen ray revealed fracture of the transverse process of the fourth lumbar vertebra and "tips of lower spines." He improved steadily, and was later discharged for limited service.

TABLE 3.—Peripheral Nerve Injuries

Nerves Injured	No. Cases	Percentage, Approximately
Median	41	15
Ulnar	45	16
Musculospiral	47	17.5
Median and posterior interosseous	1	
Median and musculospiral	6	
Internal cutaneous	2	
Musculocutaneous (upper)	4	
Circumflex	3	
Brachial plexus	20	
Sciatic trunk	33	12.5
External poplitcal	39	14.5
Internal popliteal	1	
Internal saphenous	4	
Anterior crural	6	
Small sciatic	1	
Anterial tibial	4	
Median and ulna	7	
Sciatic trunk and internal saphenous	1	
Idiohypogastric and ilio-inguinal	1	

Case 3.—Pte. C., a stretcher patient with a gunshot wound of the lumbosacral region, had a flaccid paralysis of both lower extremities with definite history of incontinence. (The first day in hospital it was necessary to catheterize the patient.) Examination revealed the left knee jerk absent, right knee jerk sluggish, both ankle jerks absent, no response to plantar stimulation, complete anesthesia to all sensation on the right side over areas corresponding to the distribution of the lower lumbar and entire sacral segments. On the left side there was a similar distribution of hypothesia. Within forty-eight hours after admission he began to improve, the first objective improvement being return of the right knee jerk. Improvement, motor and sensory, progressed rapidly but seven days after admission he developeed

double streptococcic pneumonia and died within twenty-four hours. On account of location and nature of the wound lumbar puncture was not performed, but necropsy revealed, in addition to the pulmonary findings, a machine gun bullet lodged against the fourth lumbar spine. The spinal cord and its membranes in situ and on section showed no macroscopic abnormality. There was no evidence of extravasation of blood within the spinal canal.

The total number of these injuries was 266.

The peripheral nerve injuries include all grades from slight irritative to complete. Often on admission a given nerve would have every evidence of a complete lesion, but in the course of a week or ten days regenerative phenomena would appear and proceed to partial or complete recovery. The time of appearance of regenerative phenomena was so constant—from one to two weeks after injury—that it is my opinion that one month is sufficient time for watchful waiting and that surgical intervention is then indicated. It was a common occurrence in thigh injuries to have the pitcure of sciatic trunk injury prove, in a week or ten days, to be isolated external popliteal injury. Causalgia was most distressing in the median injuries, to a less degree in the external popliteal. Ulnar causalgia promptly responded to treatment. The most common description of the pain was "It is so hot, burns like fire." It was quite common to see a soldier with hand enveloped in a cloth which he frequently moistened with cold water.

In considering the prognosis of peripheral nerve injuries, muscle tone proved so vastly superior to other signs that I practically relied on it, abandoning electricity. In this connection I might also state that I abandoned the use of electricity in the treatment of irritated, concussed and compressed nerves, relying on massage and passive movements. The occurrence and rapidity of regeneration of nerves was in the following order: Ulnar, musculospiral, anterior tibula, external popliteal, median, sciatic trunk. Peripheral nerve injuries occurred in approximately 6 per cent. of the extremity wounds.

NEUROSES

Total number of these cases was 103.

TABLE 4.—Cases of Neuroses

Hysteria	37
Neurasthenia	15
Psychasthenia	5
Unclassified psychoneuroses and constitutional psychopathic states	46

The hysterias include the monoplegias, paraplegias, "gross shakers," "nodders," mutisms and other forms of conversion. The neurasthenias include those neurasthenic states commonly seen in civil life, a large proportion of which gave a fairly definite history of having been concussed. I saw but one case of real neurasthenia. The psychasthenias all showed actual obsession. The other psychoneuroses and constitutional psychopathic states were the emotional, unstable, apprehensive, egotistical personalities. The term "gross shaker" is used to designate those cases showing gross rhythmical tremors of the entire body, not unlike that observed in astasia abasia, whereas the term "nodder" applies to those with atypical torticollis-like movement of the head and neck. Fine rhythmic tremors were demonstrable in all the psychoneurotic as well as in the neurasthenic patients. The usual exaggeration of deep reflexes was present. I saw no case of complete hemianesthesia, the affected extremity alone being anesthetic. Contraction of the visual field was rare. Of the total number of functional cases, two only had received actual wounds. One patient, a mental defective, was a "gross shaker" whose wound necessitated amputation at the thigh. His "shakes" disappeared long before the wound healed. The other patient developed a hysterical monoplegia after his wound had healed.

Besides the elements of fear and desire in the development of the neuroses, I am convinced that there were two factors which played a potent part in our cases; namely, physical exhaustion and the suggestion offered by the notoriety given so-called "shell shock" by the press of the United States. One of the phenomena of fatigue is tremor, and it was in the "shakers" or tremor cases that exhaustion was especially operative, the tremor developing as the result of exhaustion. Then, the soldier being too slowly evacuated, sufficient time elapsed for the elements of fear and desire to come into action, producing exaggeration and more or less chronicity. The "nodding" may have been, and probably was, a defensive reaction. Nevertheless, the subsequent events are at least strong, presumptive evidence that the element of exhaustion was the fundamental starting point of the existing condition.

The baneful influence of press notoriety was demonstrated in the psychoneurotic and constitutional psychopath. A large proportion of these patients always knew the nature of their trouble. It was "shell shock," and if perchance someone had unthinkingly written the words "shell shock" on the diagnosis tag or field card, it was doubly "shell shock." Of course, in these cases the element of fear or desire could be elicited, but my contention is that the suggestion offered by the stories of "shell shock" was a most fertile field for the development of this fear or desire. These were the emotional, anxiety states. The soldier would frankly say that he was all wrought up; he wanted to go back, but didn't know whether or not he could stand it; he was afraid he would go to pieces, etc., and in his behalf I must say I had every reason to believe he was sincere. He had read of the ravages of "shell shock," which enhanced his fear.

A few cases of anxiety states seemed to be of endocrine origin as shown by classical tremors, von Graef, tachycardia and the various skin reactions with, in some instances, more or less enlargement of the thyroid gland. These cases rapidly improved on rest in bed, but on any stress or exertion the symptoms would return.

In fully 90 per cent. of the functional cases there existed an underlying predisposition. It was in the exhaustion cases that this predisposition could not be elicited. The large majority of patients developing hysterical palsies gave a definite history of previous episodes. The usual story of the aphonic or mute, was that he had been gassed. Here the element of exhaustion apparently was insignificant while that of fear or desire could usually be elicited.

PSYCHOSES

These cases presented nothing out of the ordinary. Dementia praecox predominated. It will be observed that no case of delirium tremens was seen. Nor was there any case of abstinence symptoms as the result of drug addiction.

TABLE 5.—Cases of Psychoses

Dementia praecox	13
Manic depressive	6
Exhaustion	4

MENTAL DEFECTIVES

The number of mental defectives is given as ten. Undoubtedly there were more. This number does not include the total number examined for possible defective mentality, the fact being that equally as many, if not more, were found to be in reality not mental defectives, but uneducated boys, and this was particularly true of those coming from a certain section of the United States.

No résumé would be complete without acknowledging the hearty cooperation extended the neuropsychiatric department of the Center by the commanding officer of the Center, Col. George S. Skinner, M. C., and the assistance given by the individual unit commanders. I wish to express my warmest appreciation of the untiring cooperation and assistance rendered by the neuropsychiatrist of the individual hospital units whose efforts made for neurology and psychiatry a most creditable showing in the Hospital Center Mar-sur-Allier.

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Abstracts from Current Literature

A CONTRIBUTION TO THE EXPERIMENTAL STUDY OF THE LESIONS OF SHOCK. A. Mairet and G. Durante, Rev. neurol. **26**:97, 1919.

During the late war, the attention of neurologists was focused on accidents resulting from the detonation of powerful explosives, at short distances, without producing signs of external injury. The polymorphism of the lesions and the great variety of symptoms encountered in these conditions, as well as their pathogenesis, has given rise to many conflicting opinions.

Some observers consider the syndrome of shock to be purely functional and entirely different from the syndrome of emotion. Others believe both of these syndromes to be due to organic lesions, the exact nature of which is still to be determined. The lack of sufficient pathologic material has been the cause of contradictory opinions, and has led Mairet and Durante to expose a number of rabbits to powerful explosions at short distances, under conditions as nearly as possible like those met with in actual trench warfare, and to study the effects of such explosions.

In the first series of experiments (February, 1917) they placed rabbits in a cage on the ground, at the same level with the explosive, at distances varying from 1 to 2 meters. The explosives consisted of 135 gm. of melinite, which was later increased to 1,800 gm. of chedite and melinite.

Explosions with the lighter charges, in the open air, had no effect on the animals, but those with the heavier charges, and coming from a ditch, were much more efficacious. One of the animals became very weak, it fell down and lay in the cage, holding its head at first on the right and later on the left side. Examination revealed anesthesia of the cornea and extremities; it died thirty minutes later, but showed no gross lesions of the nervous system. The surviving animals seemed to suffer from dyspnea, which was temporary only and appeared to be due probably to the inhalation of toxic fumes from the explosives.

In another series of experiments (March, 1917) the arrangement was such that the explosions always occurred on the ground, the animals being at a distance of 2.5 meters from the charge, and suspended so that they received the shocks of the explosions at angles varying from 20, 30, 40 and 50 degrees.

In the last series of experiments (June, 1917) the authors utilized cross-trenches modeled exactly like those at the front. The explosives were placed on an iron bar attached to the upper opening of the trench. The animals were on top of the parapet (like a man in the reclining posture) or on a bench (like a man sitting); their position was such that they would receive the effects of the explosion directly, and at the same time would not be injured, in case they fell down.

Of eighteen animals experimented on, three were hit by fragments of explosives and were excluded from further study. Among the remaining fifteen, one was found to be dyspneic; some of them appeared shocked and excited, others feeble and depressed. Nearly all of them were breathing rapidly, for almost half an hour after the explosion. Most of them showed a temporary myosis. The final outcome was out of proportion to the apparent gravity of the early symptoms of shock. Some of the animals remained on their backs motionless, with their paws in extension, but they gradually recovered from these symptoms and at the end of twenty-four hours seemed to be as alert and as contented as they had been prior to the experiment. One rabbit, which seemed to be affected very little immediately after the explosion, died at the end of twenty-four hours. None of the remaining animals showed any anesthesia, atrophies or paresis.

Early Lesions: Macroscopically, there was little difference in the lesions of the animals that died spontaneously and those that were killed a few days later. In all cases except one, the lungs were streaked with confluent foci of congestion involving one half or two thirds of each lobe. With the exception of one case of punctiform lacerations of the anterior border of the liver, due to a direct blow with a piece of explosive, the other viscera appeared normal.

The spinal cord presented slight hemorrhagic areas irregularly distributed on its surface, with a few small blood clots adherent to the roots. The brain appeared normal in some cases, and in others there were found small meningeal hemorrhages scattered on the upper surface of the cortex or in the region of the pons.

In one case there was a large cervical subcutaneous and submuscular ecchymosis.

The histologic lesions were quite uniform. In the lungs there were foci in which the alveoli were filled with blood, and the capillaries at the margins of these foci were markedly dilated. In two cases the kidneys showed hemorrhages along the external sheaths of the blood vessels; these were apparently due to the rupture of the small arterioles in the arched arteries. The uriniferous tubules and the glomeruli were normal. The liver, except in the case previously mentioned, was free from ecchymoses.

The spinal cord presented at one point or another slight hemorrhages in the pia. Hemorrhages in the roots, however, were common and more marked; they involved the interstitial tissue between the nerve fascicles, the latter remaining practically normal. Both the anterior and posterior roots shared in this involvement, which was always confined to their free or floating portions. The ganglions were normal. In the cord itself the vessels between the white fascicles were frequently surrounded by rather extensive hemorrhagic sheaths; these were rarely seen in the gray commissure or in the anterior horns. The motor and sensory cells were normal. The peripheral portion of the white fascicles did not always stain well with the Pal method. The nerve fibers appeared irregular and distended; the myelin was disintegrated and had lost its continuity of form. The axis cylinders were occasionally absent, always irregular, curled up and moniliform (beaded); they were frequently cut up, sometimes only punctiform in size, but occasionally extremely large. The lesions extend from the periphery to the center, and gradually disappear without any sharp line of demarcation. Acute primary degeneration, as described by Claude and Lhermitte, was observed in only two animals.

The lesions in the brain were somewhat similar to those in the cord, except that the superficial hemorrhages were less marked. These were confined mostly to the middle zone of the gray matter, where normally the vessels are surrounded by lymphatic sheaths, sufficiently large to separate the neighboring nervous elements; these sheaths, having been distended with blood after a vascular rupture, keep the blood confined within them, thus preventing compression of the surrounding tissues allowing them to remain normal. Similar lesions were found in the pons and medulla. The central gray nuclei rarely showed any lesion whatever. Contrary to Mott's observation, the cells in the cortex and in the gray nuclei were perfectly normal. Finally, these authors observed circumscribed bruises on the surface of the cortex, with minute osseous fragments, which seemed to have been detached from the internal table of the bones of the skull. Were it not that they encountered the same lesions in the animals in which they studied the secondary effects of the explosions—"the late lesions"—they would have been inclined to attribute these to accidents at the necropsy.

Late Lesions: The animals intended for the study of these lesions were kept five, eight and nine months after they had been shocked. They were examined at frequent intervals and were found to be perfectly well, and without atrophy, paralyses or anesthesia.

Macroscopically they showed no evidences of any lesions in the cranium or nervous system. Histologic examination of the cord showed neither focal lesions, islands of necrosis nor cicatrices. The anterior horns, however, were irregular at the different levels; some were shorter and narrower than others. The number of motor cells was diminished and many of them were small without branched processes (dendrites). The lesions were diffuse and not destructive in nature, thus explaining the absence of paralysis and amyotrophy. There was no evidence of secondary degeneration of the white fascicles.

The Brain: In two of the animals there were seen numerous rounded tuberosities extending from the surface of the cortex to the angle of Bechterew's layer. These were lined with an incomplete layer of fibrous tissue and consisted of poorly stained, clotted material without nuclei; within this substance numerous vessels with thickened walls could be seen. The whole appearance of this lesion was that of a small cortical necrosis, into which the meningeal vessels, later, so to speak, included themselves.

Bechterew's layer and the layer of motor cells were riddled with vacuoles of various dimensions, giving the tissue an edematous appearance. The cells themselves were atrophied. These areas of vacuolar edema were not sharply outlined.

The motor cells were arranged in radiating columns, between which there were spaces free from cells. This arrangement of columns is due to a destruction of a certain number of radiating capillaries, which destroyed the cells depending on them for their blood supply. In some areas the changes in the motor cells were more marked; the cells were not distinct, they were arranged without order, and were often atrophied with small groups of well preserved cells interspersed between them. Localized meningeal adhesions, making the diffuse vascular changes more prominent, were also in evidence.

In a number of cases there were many areas filled with tracks of sclerosed neuroglia; these tracks were in the form of irregular bands or triangular spindles, which began at the cortex and extended to the zone of motor cells but did not reach the gray matter.

In two animals there were found, in a good many areas, perpendicular linear cicatrices breaking up the "geological" strata of the cortex. The authors think that the cicatrization is due to the passage of microscopic fragments of bone from the internal table of the skull. These foreign bodies are surrounded by tissue consisting of fusiform cells, arranged in the form of curly bands, and may occasionally be seen in the deeper subjacent white matter.

After reviewing in great detail the various mechanisms which have been called on to explain the pathogenesis of the lesions of shock, the authors express their belief that whereas the gross focal lesions give evidence that they are due to an actual contusion by the volume of gas resulting from an explosion, giving rise to bruising of tissue, the diffuse lesions of true shock are due to the effect of severe vibration, i. e., a general shaking up of the body; the selection of the organs involved depends on their anatomic and physiologic ability to withstand the phenomena of vibration.

In their conclusions, the authors insist on the necessity of reserving the term "shock" to the pathologic states which result from vibratory concussion or shaking up following the detonation of powerful explosives, the lesions of which are elementary, diffuse and without destruction or bruising of tissue.

The term "contusion" they would apply to states produced by having been struck by any agent, solid or gaseous, and the lesions of which are focal with bruising of tissues. The term "emotion" they apply to states due to exhaustion and over-exertion, purely functional conditions—no organic lesions.

They also call attention to the similarity in the nature and variety of the symptoms of the later lesions to those seen during the first days after a shock. The persisting sequelae and later complications are sufficiently frequent to darken the prognosis of shock even in the simplest cases. Their experiments showed that a lesion which may be simply vascular at the beginning of the disease, may be the starting point of a secondary, grave and important disability.

In considering the value of the above work, it must be remembered that it is not always possible to draw a comparison between effects of concussion on rabbits and human beings. The skeletal system of a rabbit is so frail when compared to that of man that it is necessary to take into account the force used in making experiments, and this is an extremely difficult matter to determine with any degree of scientific accuracy.

Keschner, New York.

LUMBAR PUNCTURE AS A FACTOR IN THE CAUSATION OF MENINGITIS. Paul Wegeforth, M.D., Am. J. M. Sc. **158**:183 (Aug.) 1919.

During the past year the Army Neurological School has reported results of studies made on experimental meningitis. The report showed that lumbar puncture performed on animals which had been given an intravenous injection of an organism which possessed high pathogenicity for the meninges produced a fatal meningitis.

Wegeforth and Latham attempt to show the bearing of this work on lumbar puncture performed in human beings suffering from septicemia. They performed lumbar punctures on ninety-three patients who were suspected of having meningitis. A positive diagnosis of meningitis was made in thirty-eight instances at the first puncture, and fifty-five yielded spinal fluids which contained neither pus nor organisms. Among these were six cases which at the time of the puncture had yielded positive blood cultures, three being meningococcus and three pneumococcus septicemia. Five of these cases subsequently developed meningitis—one, a pneumococcus case, recovered.

They state there were four cases of pneumococcus meningitis, and that two of these cases developed a meningitis after the withdrawal of a clear normal fluid. One of them had rigidity of the neck, and was stuporous at the time of the first puncture. Seven days later the second puncture yielded a negative fluid. Two days after this puncture there was definite rigidity of the neck and marked Kernig's sign, and lumbar puncture performed at this time yielded a cloudy fluid containing 1,200 leukocytes per c.mm., increased globulin per content, and the pneumococcus was present in both smear and culture.

The question that naturally arises in discussing a case of this kind is whether the meningitis was present at the time of the first puncture. It is noted that there was some rigidity of the neck—which proved that there was probably some irritation of the meninges at that time. If the clinical symptoms had not suggested the probability of a meningitis, lumbar puncture would not have been performed. A negative spinal fluid does not necessarily indicate the absence of an inflammatory process in the meninges. It has also been clearly established that there may be small microscopic foci of bacteria in the meninges which give no evidence of their presence either in the spinal fluid nor even macroscopically in the meninges after death, and that only careful microscopic examination of the meninges will reveal their presence. Liebermeister has stated that "in 25 per cent. of the cases of pneumonia there is distinct suppurative inflammation in the membranes of the spinal cord, even though their gross appearance is unchanged." Therefore, the case used by the authors as illustrative of the effect of puncture in producing meningitis is certainly not an unquestioned demonstration of their thesis. The second case cited was that of a soldier being treated for pneumonia who had severe headache and an atypical rash that turned out to be acne. The combination of symptoms was so suspicious that a surgeon on the meningitis service was called in consultation. The authors state that in order to be certain of his diagnosis, the specialist ordered a lumbar puncture. The fluid was clear. A blood culture was then taken and revealed the presence of pneumococcus. The following day the patient showed, what is stated to be unmistakable, but what I would consider were more definite signs of meningitis. A second lumbar puncture was performed, and this time the spinal fluid showed the presence of meningitis pneumococcus. This case likewise, therefore, is open to the same criticism that was made on the previous case. The authors look on these two cases as indicating the possibility of the removal of spinal fluid acting as a factor facilitating the infection of the meninges from the blood stream, and even go so far as to state that in the second case the obvious purpose was to "complete the clinical study," which is not borne out by the reasons which they themselves give for the puncture. As stated above, in neither of these cases can we be certain that the meningitic process had not already commenced in the meninges before the removal of the spinal fluid.

The authors describe a number of cases of meningococcus in which there were three of the fulminating type. These three cases were of very short duration—from fifteen to twenty-four hours. There were no symptoms indicative of irritation of the central nervous system, and yet the spinal puncture revealed the presence of meningitis. In these cases the severity of the septicemia unquestionably overshadowed entirely the symptoms of meningitis, and showed clearly that a meningitis causing very slight changes in the spinal fluid and with the presence of organisms in it may pass unnoticed. One of the cases is worthy of being cited in more detail. The patient was admitted to the hospital, Oct. 2, 1918, at 5:30 p. m., complaining of general malaise, pains in the back, legs and joints, headache and sore throat. He was conscious and showed no signs of meningeal involvement. The skin was covered with ecchymotic areas. His temperature was 103 F.; pulse, 112. Blood culture demonstrated a meningococcus septicemia. Lumbar puncture performed at the same time yielded clear fluids—4 cells per c.mm. and there were no organisms in the cultures or spreads. The patient was given serum intravenously. On the morning of the following day he was markedly prostrated, but could be aroused. The neck was stiff, the reflexes were abolished. The patient died at 11:30 a. m. The pathologic examination showed the presence of an early meningitis affecting the cerebral hemispheres. The authors probably claim that this early meningitis is evidence of the action of the spinal puncture on the invasion of the meninges in a meningococcus septicemia. The weakness of such an argument lies in the fact that they cannot assert in the light of our present knowledge that this early meningitis was not present before the spinal puncture, even though no organisms were found in the fluid. In the second case they reported, that of a soldier, E. A., who died twenty-four hours after the onset of his illness, and in whom no lumbar puncture was performed during life, a specimen of fluid obtained three hours post-mortem revealed a colony of gram-negative diplococci among the contaminating organisms. They were not able to grow this subculture sufficiently to type it. No gross lesion of the central nervous system was seen, but the histologic finding of the meninges showed an infiltration with large mononuclear cells. In this case it is evident that there was a beginning meningitis, even though there were no clinical signs, and even though the fluid showed only one gram-negative coccus. Had this case been punctured as had the other two, and a negative fluid obtained, and had a subsequent puncture performed either a few hours or days later revealed a positive result, the authors would probably have claimed that the puncture was the cause of the invasion of the meninges, whereas the beginning of this meningitis was present probably shortly after the onset of the infection.

They also describe a case of septicemia which recovered without showing meningeal involvement. This patient had convalesced from pneumonia, and had had a normal temperature for five days, when it suddenly rose to 103 F., and a morbilliform rash appeared. A blood culture taken revealed a meningococcus septicemia, but twelve hours later the temperature and pulse were normal, and a second blood culture taken was sterile. Lumbar puncture was not performed, and the patient recovered. This case, of course, cannot be used as an argument against lumbar puncture, because there were no signs of meningitis present, and therefore despite the meningococcus septicemia, there was no indication for the performance of a lumbar puncture. The reviewer, however, thinks that had a lumbar puncture been performed, it would have made no difference in the course of the disease, since the patient's natural resistance to the meningococcus was evidently either extremely high, or the pathogenicity of the meningococcus was extremely low.

The authors report a case of meningococcus septicemia, showing no clinical signs of meningitis, in which a lumbar puncture was made after the patient had received serum treatment. The spinal fluid had a slight blood contamination, and a blood-agar plate showed a single colony of meningococcus. The patient did not develop meningitis. The authors consider the presence of the bacterial colony due to blood contamination, and yet they state that the meninges in this instance could hardly have escaped infection "for if the organism were not already present in the subarachnoid space at the time of the puncture, there was a possibility of indirect and direct invasion from the blood stream." They account for the absence of the meningitis by assuming that the organisms had already lost their virulence, probably as a result of the intravenous serum therapy.

They cite two cases in which as soon as the clinical diagnosis of meningococcus septicemia was made serum was given intravenously, and within twelve hours specimens of blood and spinal fluid failed to show the presence of organisms. Both of these patients recovered without developing meningitis. Since no puncture was made before the intravenous injection was given, however, there is no way to determine whether or not the meninges were involved.

Two patients with meningococcus septicemia were punctured during the period of blood stream invasion, before serum was administered. The first was recovering from pneumonia, and had a normal temperature for sixteen days; it then rose to 103. He had slight headache and pain in his legs, but no other symptoms of possible meningeal involvement. The following day petechiae appeared and there was a slight Kernig's sign. Blood cultures at this time showed meningococcus, Type IV, and lumbar puncture revealed thirteen white cells and 600 red cells per c.mm. No organisms were obtained. Intravenous treatment was started and on the second day symptoms of meningeal irritation were marked, and the second lumbar puncture showed a cloudy fluid with numerous pus cells and many intracellular and extracellular gram-negative diplococci. Intraspinal and intravenous treatment was then instituted, and the patient made an uneventful recovery. The second case was similar to the first except that the patient died. Necropsy showed a widespread meningitis. The authors admit that in both of these cases there was some blood contamination of the spinal fluid at the first puncture, and admit the possibility of direct infection of the meninges through injury of the blood vessels by the exploring needle. They are, however, inclined to disregard this possibility, but we cannot overlook it in forming a fair judgment of the matter.

Herrick and Dannenberg (J. A. M. A. **73**:1322 [Nov. 1] 1919) described the results of their studies of the cerebrospinal fluid in acute disease, and stated in reference to lumbar puncture that "Of late, much has been said and written from a laboratory point of view of the dangers of lumbar puncture. After an experience with some 5,000 lumbar punctures in a large military medical center, in all sorts of medical conditions, we are not impressed with its clinical dangers. In none of the cases tabulated did true meningitis develop. Animal experiment has indicated possible dangers, under very special conditions, but its results cannot always be wisely translated bodily into terms of human pathology. Until convincing clinical proof is at hand, the skilled clinician need have no hesitation in removing from 5 to 8 c.c. of cerebrospinal fluid through a small needle, drop by drop."

In their conclusion, Wegeforth and Latham state that infection of the meninges occurs not infrequently following the release of normal spinal fluid following the infection of septicemia. They justify five of their observations with this contention. However, a close study of these five cases will convince any one that their conclusions are not justified. They recommend the following:

1. "That careful consideration be given the bacteriological study of the blood before such punctures are attempted." No one would deny the importance of such a study, not only before a lumbar puncture, but in any case where septicemia is suspected.

2. "That in acute diseases, in the absence of definite signs of irritation of the central nervous system, lumbar puncture should be avoided unless it is first conclusively shown that the blood stream is free of infection." No clinician would think of doing a lumbar puncture whether the blood is free from infection or not if there were no definite symptoms of irritation of the nervous system. Among definite symptoms, however, must be included headache, pain in the limbs and slight rigidity of the neck, which the authors do not seem to recognize as symptoms of possible meningeal irritation.

3. "That when the clinical symptoms are such as to render a lumbar puncture advisable minimal quantities of fluid should be withdrawn, sufficient only to permit necessary laboratory tests to be made." The reviewer agrees with this conclusion, not because he believes the withdrawal of large quantities of fluid would facilitate the invasion of the meninges, but because it is unnecessary.

4. "That small-bore needles should be utilized in performing the operation to prevent as much as possible subsequent leakage of spinal fluid into the surrounding tissues." This conclusion is justified, not because the leakage of spinal fluid into the surrounding tissues would cause bacterial invasion of the meninges or infection of the surrounding fluid, but because the chances of trauma to vessels and the possibility of puncture headache are lessened by the use of the small needle.

In criticizing this article, and also the work of the Neurological School, the reviewer wishes to lay stress on the harmful influence which they may exert in the treatment and diagnosis of diseases of the central nervous system. It has taken us a very long time to convince both the lay public and the medical profession that in lumbar puncture we have not only a very efficient and important means of diagnosis, but also a procedure, which, intelligently used, can be regarded as absolutely harmless. Unless the profession examines the work of these various authors critically, we fear that the opposite impression will be created and that we will be handicapped not only in using the method, but what is more important, there will also be a tendency not to use this important diagnostic measure when it is indicated and when the results obtained by it cannot be obtained in any other fashion.

In conclusion, I wish to quote the last sentence of an editorial article which appeared in *The Journal of the American Medical Association* of Sept. 27, 1919: "The precautions recommended by Wegeforth and Latham are of importance in acute general infection. Nevertheless, until we have better means of differentiating between the various forms of meningitis, it would not appear advisable to delay spinal puncture unduly."

Strauss, New York.

PHYSIOPATHIC CONTRACTURES OF PARETIC EXTREMITIES CAUSED BY CRANIOCEREBRAL WOUNDS. Considerations on the Pathogenesis of these "Reflex" Disturbances. Major Lionello De Lisi, Riv. di patol. nerv. **24**: Pts. 1 and 2, March 25, 1919.

The author does not refer to the cases of cyanosed, hyperhydrotic, edematous extremities seen in the emotional war reactions, but to actual contractions. These are not like the ordinary hemiplegic contractural attitudes, but are accompanied by all the trophic-idiomuscular-vasomotor and reflex disturbances of true paralysis. The cases do not correspond to the "reflex disturbances" described by Babinski. The author recognizes the possibility that in his cases, despite the fact that the actual paralysis is caused by craniocerebral wounds, the atypical contractures may be hysterical in origin. He has never seen a case in a simple hemiplegia due to brain injury. The contracture always develops in the upper extremity, and then only if there has been an injury of the extremity beside the craniocerebral injury. In the author's case illustrating these points, a soldier suffered a monoplegia (face and right upper extremity) from a fracture of the skull in the left parietal region on June 26, 1918. Contracture began ten days later. In April, the patient sustained a small wound on the dorsum of the right hand while cutting barbed wire. He considered the wound of no importance, and did not give the history until questioned concerning the scar found on examination. The oculocardiac reflex was inverted at first. The upper right extremity was held rigidly by the side, with the hand in the position of the obstetric hand, fingers all tensely extended and adducted, with the exception of the little finger and thumb, which were flexed strongly into the palm. The fingers could be forcibly moved, but gradually resumed the contractural attitude. There was atrophy of the entire extremity, including the small muscles of the hand. Electrical reactions were normal; the lower extremity was not paretic; there were no sensory changes, with the exception of diminished stereognostic sense in the affected hand; the deep reflexes were plus in the affected extremity with pronator clonus. There was no Babinski reflex. The abdominals and the other superficials, including corneal and pharyngeal, were normal. The affected hand was cold and cyanotic and then became sweaty; the same condition, but in a less degree, appeared in the left hand, especially in cold weather. The nails of the right hand grew rapidly, and were corrugated. The blood pressure was 135 in both extremities. The bones of the right hand showed osteoporosis.

The patient was mentally seclusive, apathetic, spoke little, was diffident and depressed. When stressed by examination or otherwise, he had tachycardia, sweating of the forehead, contractured hand and tremor. There was no change in this condition after the armistice. Only after many months of electrotherapy did the patient show any improvement.

The author differentiates the attitude in this patient's contracture by pointing out that it was caused by the small muscles of the hand rather than by the long extensors and flexors which cause the deformities in true or hysterical paralysis. In none of the author's cases were peripheral types of sensation disturbed. The most constant symptoms, besides the contracture, were the thermic and trophic changes in the extremity. Many authors have reported trophic and thermic symptoms in the extremities of true hemiplegics, even diminished secretion on the paralyzed side, as tested by pilocarpin. Some believe that the actual immobility in the paretic extremity, or the loss of energy in the destroyed cortex, causes toxins to be produced which travel along the same pathways as the trophic impulses to supramedullary centers, and thus cause the trophic and thermic symptoms.

Babinski's theory of paralysis of the sympathetic vasomotor control, causing reflexly diminished circulation, and therefore weakness and contractures in these cases, is refuted by the fact that in many cases where the vasomotor and contractural symptoms occur, no paralysis existed. An emotive psychologic factor may partly explain these cases.

Referring to his illustrative case, the author believes that without doubt the small wound in the right hand received in April, was the cause of the contracture which followed the craniocerebral injury, with monoplegia in

June; but this wound did not cause reflex disturbance until the extremity had become paralyzed, and then the contracture and other signs began ten days after the cerebral injury. He is inclined to believe that the emotional factor, besides the possibility of loss of cortical vasomotor control, must be considered. The factor of imitation, the patient having before him the picture of others afflicted like himself, is also noted. The author insists that his cases are not hysteria, although he admits that the vagus signs accompanying the emotions were present in his cases, namely, cold hands, hyperhidrosis, tachycardia, dermografia, occasionally low blood pressure, loss of weight, inversion of oculocardiac reflex, muscular hyperexcitability, tremor and even exophthalmos and Von Graefe's sign. In his cases these symptoms were explained by the acute and continuous emotional shock incident to fighting. Injury occurring at a time when the vasomotor mechanism is unstable, predisposes this mechanism, no matter what the injury and regardless of the degree of paralysis. The injury may, perhaps, by endocrino-sympathetic influences cause a defect in the nervous circulatory control, favoring an intoxication of the neuromuscular apparatus, with contractures.

Osnato, New York.

FEVER IN HYSTERIA, WITH THE REPORT OF TWO CASES.

Hubert S. Howe, Neurol. Bull. **2**:137 (April) 1919.

While fever in hysteria is not at all rare, hysterical fever, that is fever of true psychogenic origin, is extremely uncommon. Its reported presence is usually looked on with suspicion, particularly so in view of recent studies on obscure focal infections. If one bears in mind the frequency of such infections and the difficulty of their detection, and the prevalence of hysteria, one can readily see how by the law of chance the two would occasionally be combined. The fact, therefore, that no cause can be found is no proof that none exists. However, too many cases have been reported by very reliable observers for one to doubt the existence of hysterical fever. Furthermore, we know that vasomotor changes can and do exist on a psychogenic basis, and theoretically we can well conceive of fever from psychic causes. So, too, the recent attempts to correlate hysteria with disturbances in the vegetative nervous system or changes in the endocrines, may help us to speculate on the possible origin of the fever. None the less, the warning cannot be made too strong not to attribute the fever to hysteria before all the possible sources have been exhaustively studied. Apparently Dr. Howe has done that in the two cases he has reported.

The first case was that of a young unmarried woman of 21. The only things worthy of note in her past history are an attack of appendicitis, for which she was operated, and swollen gums six months before the onset of the present illness. Vomiting, headache, fever and abdominal pains were the chief symptoms. The onset was sudden nine days before admission to the hospital. Physical examination was absolutely negative. The urine showed a faint trace of albumin on one occasion, but there were no casts or other pathologic findings. Blood culture, Widal's test, the test for malaria and the Wassermann test were negative. The spinal fluid showed nothing abnormal. The von Pirquet reaction was suggestive. The blood, on several examinations, gave a leukocyte count ranging from 4,000 to 10,200, the polymorphonuclears varying from 68 to 77 per cent. Cultures from the urethra were negative. Roentgen-ray examination of the chest was negative. The temperature varied from 99 to 103 F., respirations, 18-22. During her stay in the hospital, from August 25 to October 9, she had repeated attacks of unconsciousness which showed all the earmarks of hysteria and none of that of an organic condition. Her vomiting persisted, and she was transferred to another hospital unimproved.

The second case was that of a young girl of 16. The chief complaints were fever and pain over the right cheek for one and a half years; attacks of asthma for fourteen years. The past history was not particularly significant except that she had a submucous nasal operation, incision of the right ear drum, and drainage of the ethmoidal cells and right antrum. On several occasions she was treated at St. Luke's Hospital, and once operated on for supposed tuberculous peritonitis. She had retention of urine, necessitating catheterization for weeks, her temperature ranged between 100 and 105 F., but no diagnosis was made and she was finally discharged with the label, essential fever.

General physical examination by Dr. Howe revealed little that was positive, except that the chest signs were those of asthma. The roentgenogram showed a shadow of both lungs, especially of the left apex. The blood count: white cells from 10,000 to 18,000 and polymorphonuclears from 69 to 85 per cent. Blood culture, the Widal, malaria, Wassermann, spinal fluid and urine tests were negative. The von Pirquet test was positive. A special neurologic examination revealed: hybrid gait, not significant of any organic condition; loss of touch, pain, temperature and posture senses over the entire right half of the body, including tongue, gums and external auditory meatus, except over right malar bone; and bilateral corneal and pharyngeal anesthesia. The sense of smell was absent on the right side. The field of vision was concentrically contracted. Taste was absent on the right half of the tongue.

Prolonged observation tended to confirm the diagnosis of hysteria, although her high temperature persistently continued.

As the author well points out, there was no parallelism in either case between the fever and the rates of pulse and respiration. There were no concomitant fever signs, such as malaise, thirst, pains, loss of appetite, etc. The patients were as unconscious of their fever as of all other hysterical signs and symptoms. Of particular note was the fact that there was no sign of hypermetabolism which is so significant of true fever, and although the second patient gave a history of almost constant fever for eighteen months, she showed no loss of weight. In neither case was there evidence of tampering with the thermometer on the part of the patients.

From the evidence submitted, one must concede that the author has proved the presence of hysterical fever in two patients.

WECHSLER, New York.

THE USE OF ORTHOPEDIC AND PROSTHETIC APPLIANCES IN THE LATE TREATMENT OF WAR DISABILITIES. A. B. LEMESURIER, M.D., Medical Quarterly, Department of Soldiers' Civil Reestablishment, Canada **1:**179 (July) 1919.

In an illustrated article, Dr. LeMesurier describes several standard appliances that have proved of value in Canada in treating the following types of disabilities: (1) nerve injuries; (2) disabilities of the joints; (3) disabilities of the feet, and (4) amputations.

The author observes that in nerve injuries the muscles require treatment by various appliances, and that the aim of this treatment is to attain one of three objects: (1) to prevent even momentary overstretching of the paralyzed muscles,

(2) to prevent or correct deformity caused by contraction of unparalyzed opposing muscles, or (3) to hold the limb in the best position for function. He describes briefly, with illustrations, the appliances used in treating patients with musculospiral, ulnar and sciatic lesions, these nerves being most frequently injured.

Two musculospiral splints are described, which are somewhat similar to those developed in our own army hospitals at Cape May and Staten Island, but they are not as simple in construction, nor do they appear to be as comfortable or practical. In each splint a separate thumb piece is necessary to extend the thumb, and the first phalanges remain unsupported. The splints, however, appear to be quite up to standard in the function of relaxing the extensors of the wrist. Extension of the hand is secured by pressure beneath the metacarpophalangeal joints by means of a hand piece, the other two points of pressure being the wrist and the forearm.

The appliance for ulnar paralysis used in Canada is considerably more complicated than that used in this country, but appears to carry out well the requirement of flexion of the metacarpophalangeal joints (thus compensating for the unopposed action of the extensor communis digitorum). A gutter-shaped piece, in which each finger lies, is designed to produce extension of the interphalangeal joints. Such a device would be perfectly satisfactory when contractures producing marked flexion of the fingers do not occur, but some modification would probably have to be made in cases with severe fibrotic changes.

The external portion of the sciatic nerve is found to be the source of paralysis more frequently than the internal, even when the injury is above the bifurcation. The resulting foot-drop is treated at night by the use of a simple canvas and wire "L" shaped appliance to prevent the weight of the bed covers from causing plantar flexion of the foot. The author favors the use, during the day, of a steel brace, jointed opposite the ankle, with a stop which locks when 90 degrees is reached, but allowing free movement of the joint up to that angle. The brace is attached to the sole of the shoe with rivets. In paralysis involving the muscles supplied by the internal popliteal nerve, the author advocates the use of a similar brace with the ankle joint completely locked. This brace has much to recommend it, but on the neurosurgical services at Cape May and Fox Hills a small, light spring splint, which exerts a constant pressure upward on the sole of the shoe, was preferred. The author condemns the use of such splints because a certain amount of stretching of the paralyzed muscles must necessarily occur. Our experience led us to believe that complete prolonged immobilization of any joint is likely to lead to fibrous changes of a most undesirable character. It was believed that the degree of stretching permitted by the spring splint for foot-drop is not sufficient to be injurious, and is more than compensated for by the lightness, comfort and simplicity of the spring. In cases of injury involving only the internal popliteal nerve, in contradistinction to the author's practice of complete immobilization of the ankle joint, no mechanical appliances to prevent dorsiflexion were used in our neurosurgical centers.

In the remainder of the article are described: 1. Braces which may be worn by patients having flail elbow or knee joints with poor muscular control. (2) Appliances to be used when there is stiffness of the knee or elbow joint, short of bony ankylosis, where it is desired to increase movement. 3. Appliances for treatment of disabilities of the feet. 4. Prosthetic appliances to be used following amputations.

Arnett, Philadelphia.

EPIDEMIC ENCEPHALITIS. A PRELIMINARY CONSIDERATION OF SOME OF ITS PROMINENT CLINICAL AND PATHOLOGICAL MANIFESTATIONS. Frederick Tilney and Henry Alsop Riley, Neurol. Bull. **2**:106 (March) 1919.

The authors describe twenty cases of epidemic encephalitis which came under their observation in 1918. While they consider the disease an entity of unknown pathogenicity with a distinct symptomatology, they enumerate eight, and possibly nine well-defined types, namely: lethargic, cataleptic, paralysis agitans, polioencephalitic, anterior poliomyelitic, posterior poliomyelitic, epileptomaniacal, acute psychotic and infantile (or neonatorum).

The lethargic type (four cases) represents the average picture which has so frequently appeared during the epidemic of 1918 and the present year. One case came to necropsy and only the medulla, pons, mid-brain and basal ganglions showed pathologic changes, most marked in the latter. Characteristic were pial injection and small hemorrhages visible on macrotomic section Microscopically there were revealed: small hemorrhages, engorgement of vessels, round cell infiltration, mild gliosis and a few ameboid glia cells, chromatolysis and cloudy swelling, very marked in the motor cells of the affected motor cranial nuclei, some vacuolization and slight neuronophagia. Although all the changes mentioned are seen in acute poliomyelitis, their character, intensity and variety were so distinct that one could not mistake the picture.

The cataleptic type is represented by two cases, both in young girls. Both showed marked lethargy and cranial nerve palsies in addition to the "image" rigidity characteristic of catalepsy. The paralysis agitans type includes five cases of acute onset. Two cases with cranial nerve involvement but no other symptoms are designated as the polioencephalitic type. One case of typical poliomyelitis anterior with prolonged somnolence, and one of poliomyelitis posterior with objective and subjective sensory involvement of the trigeminus and second, third and fourth sacral dermatomes together with lethargy and ophthalmoplegia, represent types 5 and 6. Characteristic of the epileptomaniacal type is a patient whose illness began with hallucinosis and delusions, followed by prolonged lethargy and later by maniacal excitement. Finally, for two weeks before death, epileptiform seizures occurred several times a day, culminating in a terminal status epilepticus. Strabismus and nystagmus showed involvement of the cranial nerves. One case showed an acute psychosis with hallucinations, delusions, disorientation, etc., on top of the lethargy. Finally three cases are described in infants, from 4 to 6 weeks old, who suddenly passed into prolonged somnolence during which they developed cranial nerve palsies.

In a critical analysis of the symptoms, the authors emphasize the comparative suddenness of onset, the profound stupor, the prevalence of cranial nerve palsies, the presence of some degree of fever, the tremor, particularly of the paralysis agitans type, the profound asthenia, restlessness and occasional hypertonus. They do not consider the spinal fluid of pathologic significance. This is rather at variance with the findings of other observers who claim that a pleocytosis even up to 200 cells is frequently encountered. The prognosis is looked on as rather grave, the authors' series having given a mortality of 25 per cent. It is worth mentioning that the epidemic of 1919 (and it seems to be quite as prevalent as that of 1918) is very much milder, the reviewer not having seen one fatality in more than twenty cases.

The authors further speculate on the nature of the disease and carefully seek to distinguish it from the poliomyelitic group or the influenza epidemic, although they were not fortunate in bringing direct evidence to bear on the specificity of epidemic encephalitis. They evidently were justified in their speculations, as Strauss and Loewe (J. A. M. A. **73**:1056 [Oct. 4] 1919) have successfully isolated and cultivated an organism from nasal washings, throat cultures and the spinal fluid of patients suffering from epidemic encephalitis, transmitted the disease to rabbits, obtained it from the animals and passed it through several generations.

The very thorough description by Tilney and Riley of the various "types" of epidemic encephalitis, deserves special consideration. They have also mentioned, from a review of the literature, a number of other "types," such as polyneuritic, cerebellar ataxic, hemiplegic, diplegic, etc. While this testifies to their accuracy of observation it appears to the reviewer that it is not wise to multiply "types." The important question ultimately is: Are we dealing with a specific disease? If we are, and there seems to be no doubt about it, then the various manifestations no more deserve to be called "types" than a tumor of the brain, say a glioma, should be variously designated because it happens to take root in different parts of the brain. Obviously the symptoms or "types" or "syndromes" vary with the location of the tumor, and so will the manifestations of epidemic encephalitis depend on the site of the lesion.

But while there is justifiable fear of possible confusion from the creation of types out of a specific pathologic entity, their careful description deserves attention for another reason. The correlation of symptoms with the various sites of the lesions serves definitely to establish the functions of those parts of the brain which are as yet not clearly understood. Our knowledge of the physiology of the brain will be greatly enhanced if we shall be able to show that a pathologic lesion has given a certain symptom or group of symptoms. So, too, will careful observation further our knowledge of pathology, and one need merely allude to the acute Parkinsons which point to the possible genesis of chronic agitans.

Wechsler, New York.

SOME BIOLOGICAL EFFECTS DUE TO HIGH EXPLOSIVES. Alfred Carver and A. Dinsley, Brain **42**:113, Part 2, 1919.

Admitting the importance of the psychogenic factors in the production of war neuroses, the authors of this article believe that a certain proportion of the nervous symptoms observed in soldiers after exposure to high explosives are physiogenic, or mechanical, in origin. In order to secure evidence to support this view, a series of experiments were made on animals by subjecting them to the effects of high explosives. A brief description is given of the properties and effects of explosives. An interesting fact presented is that high explosives may be sensitized, or made more easily detonated, by being repeatedly subjected to detonations insufficient to explode them. The inference is drawn that the nervous system may be affected in a somewhat analogous manner, resulting in a neurosis based on mechanical more than on emotional factors. Depending on the results produced at different distances from the point of detonation, three zones are roughly distinguished: zone A, or the "zone of brissance," is delimited by the area of gross disruption; zone B, the zone of decompression, and zone C in which the effects of detonation are further modified. Two series of experiments on animals are described, one on fish by exploding submerged charges of explosives, and one on rats and mice in cages at different distances from the explosives. In both series of experiments the animals as in zone A, were killed by the disruptive force. In zone B most of them were stunned but eventually recovered. In zone C the effects were less marked, but excitement and irritability were noted. The mammals that were repeatedly exposed in this zone became stuporous. Dissections showed lacerations and hemorrhages in the animals which were killed, and capillary engorgement in those surviving. No histologic examinations were made. Some interesting observations are made of healthy soldiers in an "ammunition proof and demolition section" during the course of their ordinary duties. They were well sheltered, and the element of fear was minimal. Various symptoms were noted, including active tremors, increased pulse and respiration, and in a few cases, vomiting. Those most affected became less resistant to subsequent exposure. As a result of their experiments and of observations on soldiers, the authors believe that the "indirect concussion" of high explosives produces definite changes in the central nervous system, accounting for certain of the symptoms usually ascribed to psychogenic factors. Although they present no histologic evidence to support this view, their argument is very suggestive, and in line with other evidence of the same nature.

Ingham, Philadelphia

SUBTEMPORAL DECOMPRESSION FOR ACROMEGALY; PRESENTATION OF A CASE. S. P. Goodhart, Neurol. Bull. **2**:168 (April) 1919.

In the words of the author, the patient was shown "for the purpose of demonstrating the value of subtemporal decompression in certain cases of acromegaly in which pain becomes persistent and intractable, etc. It involves furthermore consideration of the indications for either simple decompression, or a procedure of more radical nature, sellar decompression, with or without effort at removal of soft tissue."

The patient's illness had lasted several years. It began with frontal and occipital headache, pains later radiating to the face and shoulders. Later there developed weakness, loss of 40 pounds in weight, polydipsia, polyuria, glycosuria and diminished libido and potency. The bony changes in the face and extremities established the diagnosis of acromegaly. Syphilis was also diagnosed, for which the patient received antisyphilitic treatment. He was bedridden because of the severe pain; opotherapy had no effect. Spinal puncture gave no relief. Right subtemporal decompression five years after the onset of the illness resulted in complete disappearance of the pain. Immediately after the operation a temporary left homonymous hemianopsia developed; later the left pupil became dilated and reacted sluggishly to light. Left ptosis and weakness of the left internal rectus developed, and a few days later a bitemporal hemianopsia. These conditions were transitory, and were explained by the author on the supposition of an intraventricular edema of the third ventricle. This is rather problematic and would hardly explain all the signs. However, the fact remains that all of the patient's symptoms cleared up after the operation, though the bony changes remained uninfluenced.

In discussing the case, the author explains how difficult it is to make a diagnosis of struma of the pituitary or enlargement of the sella despite symptoms of acromegaly or dystrophia adiposis genitalia, and points to the unreliability of the roentgen ray in determining the size of the sella. The last point is particularly well taken, and the author justly urges the frequent making of roentgenograms before making a definite statement as to the size of the sella. It may be added that stereoscopic plates should never be omitted. In this connection one may recall an article by Howe (Neurol. Bull.) containing a study and measurements of forty sellae in normal people, and showing such wide variations that one can hardly distinguish some of them from sellae abnormally enlarged. It should be added that a great deal depends on the angle at which a picture is taken. As to the difficulty of making a diagnosis of struma of the pituitary, Goodhart describes briefly a typical case of Froehlich's syndrome which on necropsy showed a tumor of the corpora albicantia. He further dilates on the frequent presence of marked pituitary symptoms without macroscopic changes, and points out that subjective symptoms may be due to a "temporary true hyperplasia of the whole gland with resulting distention of the sensitive dural envelop, or to a local edema. In the absence of objective evidence of a neoplasm or strumous growth within the sella, it would seem that for the relief of the persistent and agonizing pain the comparatively harmless surgical procedure of the subtemporal decompression should be utilized."

While there is no great objection to a subtemporal decompression (although the operation is not altogether harmless), and it certainly is preferable to a direct attack on the sella, wherever it can possibly be avoided, it is rather difficult to decide when the lesser operation is indicated, unless at least some signs of intracranial pressure accompanying the pituitary symptoms are found. However, the fact remains that Goodhart's patient obtained marked relief, and his suggestion certainly is worth serious consideration.

Wechsler, New York.

GUNSHOT WOUNDS OF THE SCALP, WITH SPECIAL REFERENCE TO THE NEUROLOGICAL SIGNS PRESENTED. Geoffrey Jefferson, Brain **42**:93, Part 2, 1919.

The author, who served as surgical specialist attached to the Fourteenth General Hospital, British Expeditionary Forces, presents a report of fifty-four cases of unselected wounds of the scalp without cranial fracture, calling particular attention to the symptoms indicating injury to the brain. Of the fifty-four cases, forty-nine presented early neurologic symptoms which were classified into two types, namely, those in which there were signs of general disturbance and concussion, and those showing evidences of definite local cerebral contusion. The general symptoms, or those due to concussion, occurred in practically all of the cases, and may be summarized as follows: disturbances of consciousness; transient unconsciousness in 25 per cent., and a dazed or stunned condition in 25 per cent. Headache was noted in 82 per cent., most frequently frontal, and was "fixed," or of maximum intensity near the wound, in 45 per cent. of the cases. Giddiness was common, but was only noted on gross changes of posture. Nausea and vomiting were less frequent, vomiting occurring in eight and nausea in thirteen cases. Tendon reflexes were increased in twenty cases, eight of which included increased arm jerks which indicated more serious cerebral injury. Local cerebral contusion was indicated when focal symptoms were present, and occurred in seventeen cases (thirteen motor and four visual). The severity of the symptoms varied from a slight unilateral increase of the tendon reflexes to a definite hemiparesis and jacksonian convulsions. Contralateral cerebral contusions were noted in four cases, all in the motor area. In most of the cases the symptoms, both general and focal, were of a transient character, but in a few slight residual symptoms remained after several months. Some of these late symptoms were ascribed to a secondary hysteria, but the evidence presented to sustain that view is superficial and unconvincing. Three of the patients of the series were trephined, in two of whom extradural hemorrhages were found in the absence of cranial fracture.

INGHAM, Philadelphia.

MENTAL AND NERVOUS CHANGES IN THE CHILDREN OF THE VOLKSSCHULEN OF TRIER, GERMANY, CAUSED BY MALNUTRITION. SMILEY BLANTON, Mental Hygiene **3**:343 (July) 1919.

Blanton presents the result of a study of the schoolchildren of Trier, one of the German Rhenish province cities, made by the department of Sanitation and U. S. Public Health Service. Six thousand five hundred pupils were surveyed. They were between the ages of 5½ and 14, and for three years they had been under the disadvantage of an inadequate diet (particularly deficient in animal proteins, butter fats and milk). Special interest attaches to the psychologic-psychiatric findings. Retardation, by which the author understands a failure to pass one or more grades, increased 7 per cent. in the "war" years 1914-1918. It is estimated that 7.39 per cent. of the entire school population was retarded, primarily by reason of malnutrition. In view of the fact that the usual promotion standards were much relaxed, this estimate is probably low. Apparently neither outspoken feeblemindedness (0.95 per cent.) nor the group including "abnormal," "nervous," conduct disorder, organic nervous diseases, neuroses and psychoses (fifty-four cases) were affected by the nutritive enfeeblement. That only two cases of neuroses are reported is rather surprising, as Trier was bombed twenty-two times and there were ninety-seven "alarms." Only 5 per cent. of the children showed speech defects although minor difficulties such as slurring, indistinctness and lisping were exceedingly common in the younger children, indicating retarded speech development which might fairly be attributed to malnutrition. Increased fatigability, inattention, slowing of comprehension, poor memory and restlessness were almost universal.

This survey is open to the same objections attending all "large scale" studies. The practical impossibility of accurate individual examination and observation, and the necessity of conducting the work through an interpreter, must be given some weight in a consideration of the findings. However, with some allowance for the possible margin of error, the results furnish a valuable indication of the effects of malnutrition on the mental health and growth of schoolchildren.

STRECKER, Philadelphia.

PARALYSIS OF THE GLOSSOPHARYNGEAL, PNEUMOGASTRIC AND SPINAL ACCESSORY NERVES WITH CEREBELLAR SYMPTOMS. W. G. SPILLER, J. Nerv. & Ment. Dis. **49**:481 (June) 1919.

Spiller reports the case of a man of 45 years of age who gradually developed lateral movements of head, difficulty in swallowing with a tendency to choke, vertigo, slow speech, lateral nystagmus, difficulty in protrusion of tongue, anesthesia of uvula and soft palate with paralysis of the palatopharyngeal muscles, but no impairment of the functions of the vocal cords. There were in addition evidences of a lesion of the internal ears with the Bárány test; intention tremor of each hand, adiadochocinesis, loss of tendon reflexes in lower limbs with incoordination of gait; the blood Wassermann test was positive, but the cerebrospinal fluid findings were negative.

These symptoms pointed to cerebellar disease and also to paralysis of the glossopharyngeal and pneumogastric nerves probably due to a syphilitic meningitis implicating these nerves at their exit from the medulla oblongata.

It would appear from the paralysis of these nerves and the integrity of the vocal cords that there is a distinct nuclear representation of the motor supply of the soft palate and larynx, and that the former receives its motor supply from the spinal accessory and the sensory supply from the pneumogastric. Spiller calls attention to the rare occurrence of isolated paralysis of the glossopharyngeal and pneumogastric portion of the spinal accessory.

Yaskin, Philadelphia.

POSTURAL TONE OF THE RECTUM, WITH SPECIAL REFERENCE TO DYSCHEZIA (RECTAL CONSTIPATION). A. F. Hurst, Seale Hayne Neurological Studies **1**:208 (April) 1919.

Hurst contributes an interesting experimental study of dyschezia (rectal constipation) from the standpoint of rectal postural tone. By means of the introduction into the colon and rectum of rubber balloons with pump and manometer attachment, the author was able to determine the pressure (in millimeters of mercury) required to produce the sensation of rectal fulness and the call to defecation. Ordinarily this occurs when the peristaltic passage of feces from the pelvic colon is reflexly excited, by taking food into an empty stomach, drinking a glass of cold water, exercise, etc. The practical deduction from Hurst's work is a logical explanation of rectal constipation, on the basis of relaxed postural tone in the muscular wall of the rectum, which results when for any reason the call to defecation is habitually neglected. Then the sequence of events is as follows: Feces accumulate in the rectum; the muscular coat becomes more relaxed and, due to the diminishing postural tone, more expulsive effort is required. The relaxed muscular coat is inadequate and therefore evacuation is never complete; feces are always present in the rectum which normally should be clear. The lumen becomes permanently enlarged; the postural tone continuously decreased; obstinate constipation and, in extreme cases, complete muscular atony results. The author's work is in accord with Sherrington's observations and extends a very fruitful field of study.

Strecker, Philadelphia.

A PATHOLOGIC REFLEX OF THE GREAT TOE: THE REFLEX OF THE SECOND PHALANX. Piers Boveri, J. Nerv. & Ment. Dis. **49**:385 (May) 1919.

Boveri presents a new sign consisting in flexion of the second phalanx of the great toe and obtained by striking the tendo achillis while the patient is in a prone position with the legs flexed at right angles to the thighs, so that the lower limbs form, by the angle of the feet and legs, the position of "Z." In normal persons there is flexion of the feet, but not of the phalanges. The pathologic sign, which is usually accompanied by loss of the Achilles jerks, is observed in lesions of the sciatic nerve, of the roots and of the spinal cord. The author cites ten cases in which the sign was observed. Boveri concludes that this sign is diagnostic of a partial lesion of the great sciatic, of the external popliteal or of their corresponding segments in the cord. The author does not offer any definite mechanism of the production of the reflex, but suggests as possibilities that it is either a result of reflection (?) or a "propagation of the stimulus which, not normally developing for the interruption of the superficial muscles of the calf, runs more deeply into the big toe."

The explanation of the cause as furnished by the author is not quite clear. There may, however, be some value to this reflex.

Yaskin, Philadelphia.

QUALIFICATIONS OF THE PSYCHIATRIC SOCIAL WORKERS. Jessie Taft, Mental Hygiene **3**:427 (July) 1919.

Taft describes the ideal psychiatric worker as one who has at her command a "working psychology which will be able to carry out in social treatment the psychiatrist's interpretation of a patient, so far as it can be done in terms of personality." This worker cannot be defined either in terms of college education or mature years, for neither necessarily guarantees the intellectual and emotional maturity which are essential. Experience in social service is not a requisite, provided there is some familiarity with simple case work. The candidate must not be too introspective about her own mental adjustment nor should she be so extroverted as to be unsophisticated in respect to the ordinary social-psychiatric problems of the community. The born worker in this field will be "a maternal sort of a person even if she is only 20." She will have worked out a satisfactory philosophy of living and her basic attitude toward life will be one of sober optimism. She will have confidence, poise, sympathetic understanding and mental flexibility.

Strecker, Philadelphia.

EXPERIENCES IN THE IMMEDIATE TREATMENT OF WAR NEUROSES. Edward A. Strecker, Am. J. of Insan. **76**:1 (July) 1919.

This is a readable and concrete account of the neuroses of the 28th division observed during the first hours or days by a psychiatrist who had to get results. The treatment consisted of hot drinks, good meals, optimism, appeal to patriotism, explanation; "trick cures" and hypnotism were avoided. This scheme was efficacious; it sent most of the men back to duty within four days, and left no damage.

Strecker concludes that to cure neuroses in civil life, to make a business of getting results, demands the removal of legal shackles and public indifference, a comprehensive plan and a new attitude of prognostic faith on the part of the physician.

Bond, Philadelphia.

POSTURAL LENGTH AND POSTURAL TONE IN HYSTERICAL AND OTHER DEFORMITIES. A. F. Hurst, Seale Hayne Neurological Studies **1**:203 (April) 1919.

Hurst makes the very instructive observation that abnormal postures produced by hysterical contractures persist after consciousness is lost in sleep or even in deep anesthesia, notwithstanding the fact that the contracture itself disappears. He believes that the abnormal position is due to a change in the postural length and tone of the muscles, and is independent of the hysteria. The argument is convincing. By the normal postural length and tone of a muscle fiber at rest is understood a position about halfway between its extreme contraction and relaxation lengths. With Sherrington's work in mind, it is easy to conceive how a contracted position maintained for some time, might readily temporarily shorten the postural length, after the contraction has been removed. An argument against the hysterical nature of such shortening lies in the fact that it is only to be corrected by voluntary and conscious reeducation on the part of the patient and is unaffected by psychotherapy. If this theory holds true, it will no longer be reasonable to regard the abnormal positions which sometimes persist as sequels to recovered organic nerve lesions, as hysterical additions. The conception also has an application to the field of orthopedics, and the normal postural length may have some weight in deciding on the best immobilizing position for a disabled limb.

STRECKER, Philadelphia.

HYSTERICAL TRISMUS AND OTHER NEUROSES OF THE JAW.

S. H. WILKINSON, Seale Hayne Neurological Studies **1**:196 (April) 1919.

Wilkinson presents an interesting group of jaw neuroses. He feels that both autosuggestion and heterosuggestion play an important part in the development of the symptoms and that various jaw disabilities which have origin in actual disease and injury, may in this way be perpetuated as hysterical symptoms. Four cases of persistent jaw tremor (one having a duration of two years), three of trismus and one each of clonic spasm and palsy are described. The ease and rapidity with which the symptoms were removed bespeak the author's expertness with psychotherapeutic technic. Exception might be taken to the statement that under powerful suggestion "quite normal people may become the victims of hysterical symptoms." Symptoms of the type shown by the author's patient, would seem to imply the existence of an hysterical make-up, and it is difficult to conceive that this may be compatible with normality.

STRECKER, Philadelphia.

THE EDUCATION OF AN EPILEPTIC.

E. PONCE RODRIGUEZ, Rev. de Psiquiatria **1**: No. 2 (October) 1918.

The author, a pedagogue and not a physician, states his theory of the service that can be rendered to an epileptic by intensive training. This service should be rendered under the supervision of the physician in charge of the case.

He reports the case of an epileptic boy, 10 years of age. He had had attacks since infancy, and was markedly defective. This was shown especially in the lack of a vocabulary; he was able to say only a few words. His attention wandered and could be held on one subject for only a few seconds. In the motor sphere there was great activity — he threw things about impulsively. In addition, there was a certain functional deficiency in the use of the hands, which was evidenced by a lack of synergic control rather than by a paresis.

The author began his treatment slowly with simple games and amusements, attempting first to train the attention, then to increase the words in the boy's vocabulary, and finally to inspire ideas.

After three years of "assiduous labor," there was considerable improvement. The vocabulary had become "notably enriched"; the attention, except before an attack, was fairly stable, and the association of ideas seemed fairly normal. The defective life was favorably influenced. He showed interest, and became enthusiastic about music.

The frequency of the epileptic crises was markedly diminished. Instead of grand attacks, appeared equivalents and frustrated attacks.

In conclusion, the author states that this type of treatment—the care taken by the pedagogue—has great advantage in that the patient, instead of becoming a chronic invalid in the hands of a well-intentioned but not understanding family, is able to develop to a large extent and become more capable of self control.

Solomon, Boston.

THE BASAL METABOLISM IN HYPOTHYROIDISM. J. H. Means, M.D., and J. C. Aub, M.D., Arch. Int. Med. **24**:404 (Oct.) 1919.

This article comprises the observation on six patients, and includes practically every clinical condition associated with hypothyroidism. The methods employed consisted briefly in the determination of the gas exchange of the recumbent subject, while in the postabsorptive condition, by means of the Benedict apparatus and the calculation of the heat production therefrom. The heat production is calculated from the oxygen absorption and the respiratory quotient obtained, with few exceptions.

The existence of lowered respiratory metabolism in hypothyroidism and the ability of the thyroid gland ingested to raise it has long been known. The metabolic rise may range anywhere from 15 to 72 per cent. The percentage of basal metabolism in hypothyroidism varies, as low as 33 per cent. below the normal for age and sexes.

The authors claim that the determination of the basal metabolism forms a sound and convenient method for governing the dose of thyroid preparation in cases of hypothyroidism far better than the clinical picture and is of value in differential diagnosis, and that the proper dosage can be far more accurately gaged by following the effect on metabolism. The aim should be to bring the patient to the normal level (about 3 to 4 grains daily) and then to find the minimum dose that will be effective (about 1 to 2 grains).

DeLong, Philadelphia.

Society Transactions

THE AMERICAN NEUROLOGICAL ASSOCIATION

Forty-Fifth Annual Meeting at Atlantic City, N. J., June 16-18, 1919

James H. McBride, M.D., *President*

1. PRESIDENTIAL ADDRESS. Dr. James H. McBride. This article appeared in the August issue, p. 149.

2. BRAIN SURGERY IN THE WAR. Dr. Harvey Cushing, no discussion. This article appeared in the November issue, p. 493.

3. PROBLEMS IN THE SURGICAL TREATMENT OF INJURIES TO THE PERIPHERAL NERVES: THE OUTLOOK FOR THE FUTURE. Dr. Charles A. Elsberg and Andrew H. Woods. This article appeared in the December issue, p. 645.

DISCUSSION

Dr. George E. Price: I was very glad to hear Dr. Elsberg say that he placed no dependence on Tinel's sign. In our work overseas Tinel's sign proved so unreliable that it was discarded as being of no practical value and at Fort McHenry, where there was more opportunity to study the sign, we came to the same conclusion. Tinel's sign has been found in cases of complete interruption of a nerve with a wide bridge of scar tissue where one would not expect to find any nerve fibers. Even after recent complete section of the nerve, Tinel's sign has been present. Of the two theories advanced in explanation by Dr. Elsberg, one of the passage of nerve fibers through the scar tissue and the other of compensation of overlapping of sensation from other nerves, I would be in favor of the last. We have an example of that occurring normally in the musculospiral. An interesting motor problem is the inability to abduct the fingers in musculospiral palsy. If, however, extension is artificially supplied by placing the hand palm down on the table, the patient is then able to perform the movements of adduction and abduction of the fingers.

Dr. Elsberg also spoke of finding two cases of complete nerve section without loss of sensation, in the musculospiral and the internal popliteal. In the cases seen overseas and at Fort McHenry, there was no instance of total section of these nerves with complete preservation of sensation. In musculospiral cases there is usually a narrow band of anesthesia on the dorsum of the thumb that is apt to be overlooked.

One thing that the war has brought out is a marked improvement in the technic of neurosurgery, particularly in suturing operations on the nerves. In this connection it is only fair to say that some of the best results I have seen have resulted from operations by men who were neither neurosurgeons nor orthopedists, but general surgeons doing the best they could under adverse conditions.

Dr. E. Sachs: This is very interesting work that Dr. Elsberg has done on the anatomy of the peripheral nerves. Similar work was reported some years ago by Stoffel who emphasized the importance of keeping this point in mind in suturing the nerves. If one studies microscopically the regeneration of a nerve and notices the devious way a nerve filament grows, one cannot help feeling that it is not so very important, in suturing the two ends of the nerve, to attempt to approximate the proximal and distal end so very carefully. The principal factor in the successful results of peripheral nerve suture is the avoidance, as far as possible, of anything that may contribute to form scar tissue between the two ends. Dean Lewis's experiments showed how far a nerve might grow out if there was no obstruction between the two ends. This, I think, is further evidence that surgeons should avoid anything that may contribute to the formation of scar tissue and the approximation of the nerve ends.

Dr. Charles A. Elsberg: All I meant to say is that it seems to me there cannot be any question that, if a nerve has a certain structure and internal pattern, the best result will be obtained by bringing that nerve with all its fibers as near as possible into the anatomic relation in which it was originally when normal. *That* should be the ideal surgery. To be sure, nature is very good and she will cause the nerve fibers to grow around all kinds of obstruction, but that is no reason why the real object of an operation should not be the bringing of the nerve into a condition as near as possible to that which existed before the injury occurred. Any argument against such a procedure, tending to show excellent results when the nerve ends were sewed together haphazard, is not evidence of the successful work of the surgeon but of the successful work of nature. The surgeon should always suture the nerve so that its ends approximate as closely as possible to the normal nerve pattern, and the closer he gets to the normal condition, the better. *That* fact should be kept clearly before us. We can expect nature to help us, but we should make conditions such that nature can help us to the maximum.

4. GUNSHOT WOUNDS OF THE HEAD AND THEIR TREATMENT. Charles H. Frazier, M.D., and S. D. Ingham, M.D. This article appears in this number, p. 17. No discussion.

5. INFECTIVE NEURONITIS AMONG TROOPS ON ACTIVE SERVICE. Foster Kennedy, M.D. This article appeared in the December issue, p. 621. No discussion.

6. ACUTE ASCENDING PARALYSIS AMONG TROOPS, WITH PATHOLOGIC FINDINGS. Louis Casamajor, M.D. This article appeared in the December issue, p. 605. No discussion.

7. EXPERIMENTAL HEMATOGENOUS MENINGITIS. James B. Ayer, M.D. No discussion.

8. SENSORY CHANGES IN LESIONS OF THE MUSCULOSPIRAL NERVE. Arthur S. Hamilton, M.D. This article is a study of the sensory changes observed in cases of injury of the musculospiral nerve seen in the neurosurgical service of the Walter Reed General Hospital, referring particularly to the area of sensory involvement as compared with the anatomic distribution of the nerve.

DISCUSSION

Dr. Lewis J. Pollock: The anatomic distribution of the sensory fibers of peripheral nerves and the extent of sensory changes following section of peripheral nerves do not coincide. The area of analgesia in lesions of peripheral nerves has for many years been known to involve an area much smaller than the sensory fibers of that nerve are believed to physiologically subserve. This has been explained on the basis of collateral innervation through adjacent nerves. The dissimilarity of sensory loss and physiologic nerve supply had been noted by Weir Mitchell who, in 1864, stated that it was a phenomenon not at that time explainable, but which showed that the knowledge which was anatomic in nature could not be used in clinical interpretations.

Recognizing the fact that the small area of analgesia and at times its total absence, as in cases of complete section of the musculospiral nerve, is due to the overlap of other nerves, it is apparent that the complete sensory distribution of a peripheral nerve is not only to the part of the skin which it physiologically subserves, but also to that part of the skin physiologically subserved by other nerves, which it, under certain conditions, supplies with some forms of sensation. Therefore, the total sensory distribution of a nerve is the physiologic distribution, plus its overlap.

It was with this problem that we concerned ourselves and the results were: For pain the overlap of the musculospiral nerve extends over the palmar surface of the thumb and the radial border of the palm to an extension of the line between the index and ring finger. On the dorsal surface of the hand, it extends to a line parallel to the radial surface of the little finger, and encroaches on the base of the ring finger. One-half phalanges of the index finger and one phalanx of the ring finger were supplied by the nerve. The overlap to temperature is somewhat less than this and to touch markedly less, so that on the palmar surface of the hand only a small border of the thumb is subserved by this nerve. The median, ulnar and musculocutaneous in the hand and the external popliteal in the leg, likewise, have considerable overlap.

The method by which this overlap was determined was: Complete lesions of the nerves innervating the hand were studied for the smallest area of the various types of sensory function which may be lost. From this study it could be determined that the remaining area of the so-called physiologic distribution of that nerve was supplied by the overlapping of other nerves and furnished an index to the limit of such supply.

If the hand is supplied by four nerves and two of these are out, it is obvious that the remaining portion of the skin in which sensation is intact must receive that sensation from the remaining two nerves. If, therefore, in a complete ulnar and median lesion, the interior surface of the hand in its radial half can feel pain, this pain must be subserved by the radial and musculocutaneous nerves. It remains only to study a combined lesion of the radial and median nerve when the extent of the musculocutaneous overlap is determined. What remains, therefore, in the various combinations of peripheral nerve lesions, certified at operation, such as ulnar and median, median and radial, ulnar, median, and radial, musculocutaneous, internal cutaneous and ulnar, etc., the complete sensory distribution of any peripheral nerve of the hand, may be determined by noting the residual sensibility.

The striking feature of this investigation is the large extent of skin which has been found to be subserved by the overlapping innervation of other nerves.

That this overlap subserves only the undifferentiated types of sensation as prick pain offers a situation which needs some consideration as to Head's theory of epicritic and protopathic sensibility. In the light of the extensive overlap to pain, as compared to touch, the theory of dual innervation must again be scrutinized. It is a fact that immediately after injury the overlapping sensory nerves do not begin to functionate until two, three or more weeks have elapsed. After the sensory function has once been taken over by the overlapping nerves, resection and suture of the injured nerve produces practically no change in this area.

Any return of sensation in the area of a possible overlap cannot be accepted as a sign of regeneration. It is entirely possible that the return to prick pain in the area not included in the overlap returns only with the sense of touch, and what was heretofore interpreted as the return of protopathic sense in a nerve, was only the assumption of function of an overlapping nerve.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Oct. 16, 1919

George A. Waterman, M.D., *President*

PUNCTURE OF CISTERNA MAGNA IN MENINGEAL BLOCK. Presented by Dr. James B. Ayer.

During the past few years, there have been brought forward, particularly by French writers, a number of methods designed to reach the cerebrospinal fluid spaces when lumbar puncture fails because of meningeal block. The principal methods employed have been puncture of the subarachnoid space at thoracic and lower cervical levels, also puncture of the basilar cisterna by introduction of the needle through the sphenoidal fissure, and puncture of the lateral ventricles. The usefulness of these procedures varies, of course, with the seat of obstruction.

In June, the writer with others (Wegeforth, Ayer and Essick, Am. J. M. Sc. **47**:789, 1919), published the technic for performing puncture of the cisterna magna, by introduction of a hollow needle through the occipito-atlantoid ligament. This procedure was found to be easy on the cadaver. By injection of fluid blackened with carbon, free communication was found to exist between the cistern and the cerebral and spinal subarachnoid spaces. This puncture was therefore advocated as a method of tapping the upper fluid reservoirs in cases of blocking at any point in the spinal canal.

The writer has recently had occasion to put cistern puncture to a practical test in such a case. The patient was first seen after meningitis had been present for about five weeks. Lumbar puncture yielded 6 c.c. only of clear yellow fluid which clotted entirely in a few minutes and which contained a large amount of proteid, only a few cells and no bacteria, either in film or culture. Puncture of the cisterna yielded 20 c.c. (more could easily have been obtained) of white purulent fluid under high pressure (400 mm. by manometer) containing a large number of polymorphonuclear leukocytes and gram cocci. On culture the organism was identified as *Staphylococcus aureus*. The lumbar fluid, presenting the Froin syndrome, together with the absence of organisms, may be considered as conclusive evidence of blockage of the spinal subarachnoid space at some point; the cistern fluid unquestionably drained infected cerebral meninges.

Two subsequent cistern punctures have been performed in this case, the fluid on the second occasion being much less cloudy and on the third almost clear, with few cells and no bacteria in film or culture. In spite of this fact, however, the patient now presents symptoms probably referable to internal hydrocephalus, and it is likely that ventricular infection still persists, for which puncture of the lateral ventricles will be required.

The case is presented because it clearly shows the availability of cistern puncture in spinal subarachnoid block. The puncture was performed with ease on all three occasions, and with no ill effects to the patient at the time or subsequently. On the first occasion ether was given; the other two punctures were made under procain anesthesia.

MEDITATIONS ON MORALE. Presented by Dr. Donald Gregg.

Dr. Gregg defined moràle as a state of mind or consciousness conducive to the control of conduct by intellectual ideals rather than by instinctive impulses, and pointed out the various instincts which have been studied in their effect on human conduct as expressed by Bergson, Jung, Adler, Freud, Trotter and Sidis. He brought out the fact that two fundamental instincts, the gregarious and the self-preservative, have been of especial importance during the war in relation to morale. Inheritance, acquired disabilities and environmental strain he regarded as the factors conducive to the augmentation of the instinct and reactions, and pointed out how each of these played a definite part in individual development. He spoke further of the morale division of the army whose chief effort had been to augment intellectual ideas and to remove or mitigate as far as possible conditions that beget ill health, anxiety and fear.

If we could plot out our intellectual ideas and our instincts, Dr. Gregg queries whether we would not find that our morale would be represented by the separation between the two curves, and whether with a fall in ideals or with a rise of instinctive reactions the margin of morale would not decrease. He concludes that without morale an army is ready for defeat and rout, and an individual is ready to develop psychoneurosis.

MEDICAL AND SOCIAL ANALYSIS OF DATA OF OUTPATIENT DEPARTMENT, PSYCHOPATHIC DEPARTMENT, BOSTON STATE HOSPITAL. Presented by Dr. A. Myerson.

Psychiatry was born in the asylum and up to the present time has carried the point of view therein engendered. The central conception of insanity which is essentially a legal matter has tremendously hampered the psychiatrist, nor does the substitution of the word psychosis help matters. The outpatient psychiatrist takes this point of view: he deals with mental disease and includes in this category on an even basis the so-called psychoses and the so-called psychoneuroses. Concerning the former, it may be stated that not every dementia praecox patient is insane, nor is every patient afflicted with general paresis insane. Concerning psychoneuroses, every case of psychasthenia and every case of hysteria is a purely mental disease, and the neurosis part of the title is a shadowy euphemism. Some psychoneurotics need to be committed to an insane hospital though the majority do not. Commitment is to be regarded as a social and individual therapeutic measure, and has no bearing on diagnosis or classification. All cases coming before the psychiatrist and diagnosed as mental disease bring up the question of therapeutics, and among these commitment is only one of the therapeutic possibilities in psychiatry. It may be necessary to utilize it in a case of dementia praecox, and it may be necessary to utilize it in a case of anxiety neurosis.

An effort was made to analyze the statistics of the outpatient psychopathic department of the Boston State Hospital with the following questions in mind: 1. Who sent the patient? 2. What problem does he present? 3. What diagnosis is made? 4. What is done with him? The answers to these questions throw light on the social and psychiatric problems of the community.

The largest individual group of cases came from social agencies, and their interest was greatest in the female adolescent; next, in the female adult; male children were third in importance; female children fourth, and the male adolescents were given scant attention in so far as their mental state is concerned. The principal problems brought for solution by the social agencies were delinquency, especially of the sex type and illegitimate maternity. Of the large variety of diagnoses made in the social cases, mental deficiency took first rank in point of numbers. Psychoneuroses were relatively uncommon and various psychoses occurred fairly frequently. A large percentage of the cases had no disease that could be diagnosed; in other words, were merely suspects. Interesting relationships were found in the case of other sources from which the patients came, such as the courts, schools, positions, other hospitals, own initiative and the psychopathic department wards. Each source of patients was individual in the type of cases sent and in the problems presented. The psychopathic outpatient department is a consulting house for diversified groups of people, each one of whom is individual in the problem presented.

HISTORICAL CAUSES OF THE PSYCHONEUROSES OF THE JEW.

Presented by Dr. A. Myerson.

Among various matters considered important in the problems presented in neuropsychiatric clinics is the preponderance of Jewish psychoneurotics. The historical background of the Jew was searched for causes, as has often been done before. It is contended that before biologic heredity can be evoked as a cause of racial predisposition and liability to disease, what is termed social heredity must first be analyzed and examined. Social heredity is a group of life conditions and opinions handed down from generation to generation and of extraordinary potency in molding the character of a race.

In the case of the Jew this may be briefly summarized as follows: Owing to the restrictions and limitations made by his Christian "hosts" he was excluded from the soil and debarred from handicraftsmanship. Thus his manual motor instincts were suppressed. The large basal movements by which psysique is built up and nervous tension discharged were made inaccessible and he became almost exclusively an urban dweller, excessively cerebral in his pursuits, despising exercise and sports, and thereby lowered his physique and endurance. Due to the precariousness of his social and economic position, the threshold for fear was lowered; due to the close family life and the close racial life, an unhealthy emotionality was engendered, though this family life and clandishness in part accounted for the survival of the Jew. Introspection came easily to a race of cerebral workers, excluded from sports and very apprehensive. These factors may explain the psychoneuroses of the Jew.

That social heredity is easily cast off under freer conditions may be seen at the present time in Jewry. The close and pure family life is becoming a thing of the past. Antipathy to sports and the poor physique therein resulting, tends to disappear in the second generation. The Jewish prize fighter is as common and as successful as the Jewish scholar. The overvaluation of money can hardly be exemplified in these sons of the pious Jew who now are the greatest sports in America, and obnoxiously so. Social heredity differs from biologic heredity in the quick changes that may occur once the link with the past is broken, and this seems to be occurring at the present time in the case of the American Jew.

DISCUSSION

Dr. Donald Gregg asked whether Dr. Myerson had any theory as to the basis of the instinctive dislike of one race for another. That is to say, inasmuch as various races have varied immunities to disease, there may very possibly be an instinctive objection to another race on a biologic basis, if that race is subject to certain diseases that the first race is not. For instance, the negro has a different immunity to tuberculosis and syphilis from the Anglo-Saxon. Is it possible that the instinctive feeling that the Anglo-Saxon has toward the negro is based on such biologic facts?

Dr. E. W. Taylor asked Dr. Myerson whether there were any statistics or facts available relative to the part the Jews had taken in the war. Of course, they entered the war in large numbers and his impression was that they stood up under it equally with any other group, or class or race.

He spoke in strong commendation of Dr. Myerson's opening remarks on the use of terms, particularly on the use of the term insanity and especially the confusion of the legal with the medical aspect of the term and agreed with him that the term psychoneurosis is a misnomer, that the neurotic element implying structural change is tremendously in abeyance and is nonexistent in a great many instances. Hysteria, for example, is a true psychosis. It is, however, useless to enter into a discussion as to the terminology as this is one of the greatest difficulties in clarifying medicine in general and neuropsychiatry in particular. The point he would make was that it was desirable to study each case carefully and to use terms as far as possible in a general rather than in a too specific way.

It is regrettable that there is a feeling on the part of the community that the psychopathic hospital is still a hospital for the insane. However, the outpatient department has done, and is doing, a great deal to overcome this feeling and all the agencies, physicians included, should be more generous in sending patients to the psychopathic hospital in order, among other things, as far as possible to overcome this still prevalent feeling. The greatest possible advance in the last few years has been to recognize the predominant mental character of a great number of the conditions dealt with.

Dr. Walter Channing expressed himself as delighted with Dr. Myerson's paper in that he had summarized the great advance that has been made in the last ten years in the consideration and treatment of the whole subject of mental diseases. We probably can never have an exact classification of such diseases because the mental states are really without number, and one may to a certain extent run into another so that we can only in a general way describe them. Every time we hear any one speak of the use of the word insanity as did Dr. Myerson and Dr. Taylor, we are making progress. The word has become merely a social term, meaning that a person subject to mental disease is regarded as a proper case for an institution, and that it is proper to commit him. In the last analysis the most important outcome of the psychopathic hospital will be the outpatient department. A great many years ago, a little department of the Boston Dispensary was started, but patients almost had to be paid to go there. Even at that time the Jews constituted a large part of the patients. They seemed to be more intelligent and more willing to come and place themselves under treatment without undue sensitiveness about being called possible cases of insanity.

Dr. Alfred T. Noyes called attention to the fact that Dr. Myerson had, by means of tables (which were shown), presented very concrete facts as to where the patients came from, what the diagnosis was, what the problem presented and what was to be done for them. Heretofore at the psychopathic department there has not been a concrete arrangement in just this manner. Due largely to Dr. May's instigation, Dr. Myerson has evolved this scheme, and this is the first year in which it has been possible to apply it. If this is applied for say fifteen years, some very practical statistics will result.

Dr. Myerson in closing in answer to Dr. Gregg's question as to whether or not an antipathy of an instinctive kind exists in races, thought this possible. It is certainly true that perception of unlikeness in any gross way arouses an instinctive dislike. Unlikeness arouses dislike. The feeling that we are just right makes us antagonistic to one unlike ourselves. This is noticeable in children. In the movies, for example, when pictures of foreign peoples are shown a titter is apt to run through the audience. Even though in themselves pleasing, nevertheless differences in custom and costume arouse an instinctive dislike. It requires education and study to recognize that what is unlike ourselves possibly is as good as we. This is the process of education. As to whether there is an antagonism on the basis of disease, he expresed no opinion. Other far more palpable unlikenesses exist. The negro is disliked because of his black skin. The Jew is different and therefore the subject of dislike.

Dr. Taylor inquired about the army. The statistics of the army are very good. There are less than 3,000,000 jews in the United States, most of them on the Atlantic seaboard. The total population of the country is over 100,000,000; a little under 3 per cent., therefore, are Jews. The number of Jews in the army was over 4 per cent., an increase of one third over the allotment. There were a great many Jews who objected to serving. The native born Jew was as willing as any one to enter the army. The foreign born Jew, on the other hand, largely Russian, showed for a long time antipathy to to the war largely because he was at first pro-German for the reason that Russia was on the other side. Consequently, he rejoiced when the Germans beat the Russians. When the United States entered the war the situation changed. The fatalities and casualties among the Jews were as large in proportion to their numbers as those of other races.

Concerning the general subject of insanity, he thought the critical point was that we are always getting into difficulties when we make an abstraction from a reality. Insanity is an abstraction. We are dealing merely with mental disease. Given a mental disease—hysteria, psychoneurosis, dementia praecox, anything you like—it may or may not be a committable case. The larger number of general paretics become committable, but not all paretics are insane or committable. On the other hand, the majority of hysterics are not committable but there is nothing in hysteria to prevent such a step from becoming necessary; similarly with psychasthenia. Commitment is merely a legal and therapeutic step, not at all diagnostic; at present it is locked up with "insanity" and psychosis and these terms should be dropped.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Oct. 24, 1919

J. Hendrie Lloyd, M.D., *President*

CASE OF A EUNUCH CONVICTED OF RAPE. Presented by Dr. N. S. Yawger.

This presentation by Dr. Yawger showed the retention of sex desire to a felonious extent many years after complete extirpation of the gonads.

A colored man who was sexually active at 18 years sustained a severe injury by being struck by a locomotive. He was knocked astride a switch-post thereby fracturing his skull and right leg and badly lacerating both testicles; the latter were subsequently completely removed. The man lay unconscious for five days and then recovered. As a result of this injury, according to his statement, he differed from his former self only in the loss of about 30 pounds in weight, but that in particular he was absolutely without sexual power and sexual desire.

In December, 1917, a white woman charged that he had committed rape on her. The man was indicted, tried and found guilty. He was then 42 years of age. The fact that he had been castrated 24 years before was not brought out during the trial, but this condition was the prominent feature of the request for a new trial. The case was not reopened, and a sentence of from ten to fifteen years in the Eastern State Penitentiary was imposed. Shortly following the convict's entrance into the prison, an interview was sought with Dr. Yawger when the above facts were disclosed, and he repeated emphatically that following castration there had been neither sexual power nor sexual desire. The prisoner then asked, since it was impossible for him to have committed rape, whether he should not be pardoned?

Examination of the castrate showed him to be rather thin, flabby and in appearance several years older than his stated age; the penis was easily of average size. Dr. Yawger believed in regard to his sexual feeling and capacity that the prisoner had prevaricated, and told him so.

Literature bearing on this subject is interesting. Breeders of stock tell us that the physical appearance and nature of a male undergoes a vastly greater change when castrated young than when this operation is performed on the adult; and that the sex desire is especially likely to remain if the animal has previously been standing at stud. Formerly, at the earnest solicitation of parents, surgeons occasionally castrated boys afflicted with feeblemindedness and dementia praecox with the thought that masturbation would be overcome and in the hope that mental improvement would then ensue; some of these boys, on the healing of their wounds, resumed their pernicious habit and none were advanced to normal mentality. That considerable sexual power may persist in a eunuch, appears to have been recognized, since we are informed that the mutilation of males attendant on harems was frequently carried beyond castration—sometimes the penis was amputated.

A CASE SHOWING JACKSONIAN ATTACKS (STATUS EPILEPTICUS) WITH NECROPSY. Presented by Dr. Samuel Leopold.

Dr. Leopold said that epilepsy of syphilitic origin is not an infrequent picture, but this patient is of interest because she showed a status epilepticus and because there was an opportunity to study the pathologic process.

A colored woman, aged 38, twelve years ago had fits in which she would run around the house and, according to the sister, would tear off her clothes. These attacks occurred at irregular intervals until her admission to the hospital.

For several days before admission to the hospital she had had several convulsions a day. She was admitted to the hospital during one of these attacks which ceased half an hour later. The next morning, at 9 o'clock, the convulsions began again and continued constantly until she died at 11 o'clock that night. The attacks were limited to the left side of the face, left arm and leg, and were clonic in character. There was conjugate deviation of the eyes to the left, and the head was turned to the left.

The convulsions started in the face, then involved the neck, arm and leg on the left side; occasionally there was a slight movement in the right ankle and foot. The patient was unconscious; the movements were slow and coarse. The pupils were contracted and did not react to light. A study of the reflexes was not satisfactory, all seemed to be lost. Heart and lungs were negative. Wassermann reaction of the spinal fluid was positive.

At necropsy, the brain showed a marked gummatous meningoencephalitis which involved the right frontal lobe and extended to the precentral convolution.

A CASE OF ESSENTIAL EPILEPSY EXHIBITING SPECIAL FEATURES AND GREATLY IMPROVING UNDER OPERATIVE PROCEDURE. Presented by Dr. Alfred Gordon.

A male, aged 32, developed at the age of 12 epileptic seizures of a generalized character. Until six months ago the attacks occurred very frequently, from two to four daily, with short intervals of intermission, the longest of which was one week. Six months ago, when the man first came under Dr. Gordon's observation, he had had an unusually severe attack. After recovery a distinct aphasia was observed. It was of a pure motor type (aphemia) in which the inner speech was preserved. There was no paralysis of the extremities, and no indication of the involvement of the motor area or its tracts. The reflexes, deep and superficial, were unaltered.

The eye examination was negative, with the exception of irregularity of the right pupil. This, according to the ophthalmologist, was of no significance.

The examination of the urine, the Wassermann test on the blood and spinal fluid and the roentgen-ray investigation of the skull were all negative. There was no history of venereal infection.

In view of the clearly defined motor aphasia, the presumption was that a hemorrhage probably took place over Broca's convolution. On the thirteenth day following the supposed hemorrhage, the aphasia remained unaltered, contrary to what is usually observed in cases of aphasia occurring in the course of epilepsy. In the majority of instances the aphasia is temporary and transient, comparable to the paresis of one or two extremities which sometimes occur in epilepsy. These phenomena are all due to a temporary inhibition of the corresponding centers and of their tracts.

In view of the persistence of the disorder, a lesion in the form of a hemorrhage was considered. Operation was advised and promptly accepted by the patient. An osteoplastic flap was removed from the left side of the skull over the frontoparietal region. A hemorrhagic focus was found over the lowest portion of the third frontal convolution.

Rapidly the patient's speech commenced to improve, and in three or four weeks returned to almost normal. For four subsequent months there was no convulsive seizure. Due to some indiscretions in sexual matters and to an enormous meal on the same evening, he became ill and had his first convulsive seizure since the operation. His speech again became affected. During the last two months there had been no epileptic attacks. The patient showed some disturbance in speech, but no other abnormal condition. The case was interesting from several standpoints: (1) The occurrence of a hemorrhage during an epileptic attack over Broca's region producing a persistent motor aphasia. The condition was very exceptional. (2) Arrest of persistent convulsive seizures of a violent character following an operative procedure on the skull. (3) A practical question arises as to the advisability of performing a cranial decompression in essential epilepsies even without focal and well localized symptoms (that the intracranial pressure in all epilepsies is elevated, is a well-known fact).

DISCUSSION

Dr. William Spiller said that a child had been brought to the dispensary of the University Hospital recently whose condition was similar to that in the case presented. The child was about 15 years of age; it had had general convulsions most of its life, and four years ago had become hemiplegic. Dr. Spiller thought that possibly the hemiplegia had developed as a result of the convulsions, but the paralysis had been of too long duration to permit operation. He recalled a case of hemorrhage into the conjunctiva, occurring during epileptic convulsions. Where there was congestion, such as occurs in repeated epileptic attacks, there might be a rupture of a blood vessel in the brain.

The fact that Dr. Gordon's patient had had no attack for four months after the operation did not necessarily mean that the man would have no more convulsions. There may be benefit from an operation on the brain in epilepsy, for a time, even when nothing has been removed.

Dr. Spiller thought Dr. Gordon's patient did not have aphasia. His speech was very indistinct, but there was no true aphasia.

Dr. J. Hendrie Lloyd said that he recalled Dr. Gordon's case. The patient was under his care at the Philadelphia Hospital. He was surprised to see him put down as a case of essential epilepsy, with aphasia. Dr. Lloyd thought the man had some speech defect more like a dysarthria. He thought this point important because it antedated Dr. Gordon's first observation of the case. His recollection of it was that it was not an aphasia, it was more like a dysarthria. It was an interesting point and that was the reason he dwelt on it.

Dr. Alfred Gordon said that he was surprised to hear that the patient had been in the Philadelphia Hospital. At the time he saw him first, in April of this year, the history was given to him just as he had reported it, and following his last attack, which he had had a few days before Dr. Gordon saw him, he developed this aphasia. Formerly he spoke perfectly well. That was the testimony of his father, mother and sisters. Then the operation revealed the hemorrhage over the lowest portion of Broca's convolution and the ascending frontals. The operation was beneficial. The surgeon removed the blood, washed it out and the cortex appeared quite clear. The operation was performed ten or eleven days after the attack; the patient began to talk a few days later. The inhibition of the parts corresponding to the speech center persisted to a degree, but he made rapid progress, so much so that the aphasia was considered quite cured. A new attack occurred and then the speech again became disturbed. Perhaps he had a second hemorrhage. He refused a second operation, which might have improved his speech.

A CASE FOR DIAGNOSIS. Presented by Dr. S. F. Gilpin.

A white male, aged 62 years, a carpenter by occupation, fell 6 feet into the hold of a ship in November, 1918, which caused the present condition. The man was rendered unconscious, and was taken to a hospital. The roentgen-ray examination at the hospital showed a fracture of the orbital plate of the frontal bone. An operation was not performed. The patient was unconscious for at least four hours. While in the hospital he complained of pain in his arms, and he was unable to raise them or to sit alone. During this time he had incontinence of urine. He remained in the hospital sixteen days, and was taken home on a stretcher, being unable to use his arms and feet He was out of bed in five weeks, and dragged his right foot in walking. The control over the urinary sphincter returned about this time, but he lacked perfect control of his bowels. He was fed by his daughter for three months after his discharge from the hospital. A tremor developed during the past three months.

Examination, Oct. 11, 1919, showed that the pupils reacted promptly, station showed some slight increase in sway. The man arose from a chair and sat down stiffly. His gait was a shuffling, hesitating one; he scraped his right foot along the floor and had a tendency to turn toward the right. A coarse general tremor was produced when he attempted to walk; this tremor was most marked in the hands. He carried his upper extremities in the position of a case of paralysis agitans. His grip was weak in both hands, but equally so. The sphincters were controlled. All motion was slow and stiff. He assisted himself with his hands when crossing his legs. The knee jerks were rather exaggerated; there was no Babinski and no foot clonus. There was no sensory disturbance. The mental condition was normal, except that he was emotional; at times he broke down and cried. He was given compensation at the rate of $10 per week for four hundred weeks.

The case was an important one from the practical standpoint of diagnosis in cases requiring compensation. Was this a case of paralysis agitans? The patient's muscular weakness and rigidity of attitude in turning suggest this disease. However, the tremor came out on attempting motion, and was especially marked when the patient knew he was being observed. When climbing stairs he showed no tremor. If he had paralysis agitans, was the disease developing before the trauma or was the trauma the direct cause? His gait and general bearing suggested hysteria. If hysteria was the explanation of his case he should be cured and the compensation adjusted. It would seem plausible to ascribe his symptoms to the severe concussion his brain must have suffered to produce the symptoms immediately following the accident.

DISCUSSION

Dr. F. X. Dercum said that the attitude of the patient and his manner of turning around strongly suggested paralysis agitans. There was no tremor, however, except just as the hands came to rest, but tremor is occasionally absent in paralysis agitans. However, the fact that the man was a "compensation case" and was at present receiving compensation, which was to extend over a number of years, must be taken into consideration. It was found that very strange symptoms were every now and then presented by litigation and compensation cases, symptoms which did not subside until the case was settled in litigation cases or when the payments ceased in compensation cases. Of course this case was perhaps a genuine case of paralysis agitans and not one of hysteria, and it was possible also that the man may have been developing paralysis agitans when he received his fall. The weight of the evidence, however, Dr. Dercum thought, was in favor of hysteria.

Dr. J. Hendrie Lloyd said that as Dr. Gilpin raised the question of hysteria, he would say that the gait looked very much like the gait of astasia-abasia. He had, however, never seen a case of astasia-abasia in a middle-aged man; it usually occurred in young women.

Dr. William G. Spiller said astasia-abasia did occur in men of middle age, and he had recently had such a case. There have been many cases in which trauma has been supposed to be the predisposing cause of paralysis agitans.

Dr. Charles K. Mills said the patient looked to him like a man who had had paralysis agitans, and then had a traumatism which resulted in hysterical astasia-abasia.

Dr. Alfred Gordon said that this man had a fracture of the skull. It was demonstrated by the roentgenogram. If it was a question of speculation, why not consider other possibilities, such as involvement of the lenticular nucleus? In diseases of the lenticular nucleus some features of the present case have been observed. The patient may have had a hemorrhage into the lenticular nucleus by contre-coup. As far as the astasia-abasia or paralysis agitans were concerned, the patient's condition was not typical of paralysis agitans nor of astasia-abasia. Dr. Gordon said he believed in the possibility of a real organic lesion in this case.

Dr. S. F. Gilpin said that he had seen astasia-abasia in middle-aged men. The marked general tremor when the man tried to walk, and again, when he was told to control it, his ability to control it, and further, Dr. Gilpin said he had watched him climb stairs to the second floor without a sign of tremor. Evidently some of these symptoms were controlled more or less. Dr. Gilpin stated that the roentgen ray did not show fracture at the present time. He did not see why the condition in a man whose nervous system had been shocked as had this man's, should not have an organic basis.

A CASE OF POSTEROLATERAL SCLEROSIS ASSOCIATED WITH ANEMIA. Presented by Dr. Joseph McIver.

A white male, aged 53, was admitted to the service of Dr. James Hendrie Lloyd, at the Philadelphia General Hospital, September 2 of this year. Dr. McIver's principal reason for bringing the case before the society was the fact that syphilis had been suggested as a possible etiologic factor.

About April, 1918, the patient complained of an aching pain in the left shoulder, which had continued at varying intervals to the present time. Some time later he noticed paresthesias in the tips of the fingers of both hands. This symptom had continued and had steadily grown worse. In the early part of this year he noticed that his legs were becoming weak, and he had to give up his work as a laborer. Soon he began to suffer from paresthesias in the lower extremities. The condition had slowly progressed and since September 2 he had been confined to bed.

The family history was negative. The past medical history was likewise negative except for gonorrhea twenty-five years ago. He denies very emphatically any syphilitic infection. He complained of weakness and paresthesias in both lower extremities. He stated that his heels felt as though the bones were going through the flesh. There was also a stinging sensation in the hands and feet. There was a history of incontinence of feces on one occasion.

Physical Examination: The pupils showed a slight inequality, but reacted well for light and in accommodation. The eye grounds were normal. The teeth were in very bad condition. The biceps and triceps reflexes were present and normal. There was a slight degree of ataxia in the finger to nose test, but there were no sensory disturbances in either of the upper extremities.

The lower limbs were spastic, and there was a marked Romberg sign. There was marked ataxia in gait and in the heel to knee test. The achillis tendon and patellar tendon reflexes were markedly exaggerated; the Babinski reflex was present on both sides. Tactile, pain and temperature senses were normal throughout. The sense of position and vibratory sense were lost in both lower extremities. The urine was negative. An analysis of the gastric contents showed free hydrochloric acid 17, total acidity 50, and lactic acid present. The examination of feces for ova and parasites was negative. Blood Wassermann was negative. Spinal fluid Wassermann on one occasion was + + with a cholesterinized antigen; and on two occasions was anticomplementary. In discussing this report with the serologist, Dr. McIver was convinced that very little positive significance could be attached to it. Cell counts of the spinal fluid were 30, 10 and 20 on various occasions.

Blood Count: Hemoglobin, 60 per cent.; erythrocytes, 2,229,000 per cubic millimeter; leukocytes, 7,000 per cubic millimeter; color index, 1.3. Differentials: polymorphonuclears, 68 per cent.; small lymphocytes, 18 per cent.; large lymphocytes, 7 per cent.; eosinophils, 7 per cent.; basophils, none. Abnormal erythrocytes: few metachromatic, few microcytes, few macrocytes. Nucleated erythrocytes not found. Abnormal leukocytes not found except an excess of eosinophils. Blood diagnosis: secondary anemia, pernicious in type.

Dr. McIver said that in combined sclerosis of the spinal cord associated with anemia, the degeneration of the posterior column was peculiar in that it began in the more medially situated fibers, that was to say, in the long fibers of the columns of Goll. Consequently in the early stages, deep sensation alone was disturbed, more particularly, bone sensation and the sense of muscular position. In April, 1916, Dr. William G. Spiller reported to the Philadelphia Neurological Society the microscopic findings of the spinal cord in a case of tabes associated with severe anemia. He was able to demonstrate, not only the usual lesions of tabes dorsalis, but also the characteristic alterations of the spinal cord seen in anemia.

So far as Dr. McIver had been able to discover, these particular sensory phenomena did not occur as isolated signs of syphilitic disease, but were associated sooner or later with a type of anemia that was fatal.

Preservation of pain and tactile sensations with increased tendon reflexes was the rule in the early stages of combined sclerosis associated with anemia. These sensations might be affected late in the course of the disease with diminution or loss of the tendon reflexes. In cases of syphilitic posterolateral sclerosis, pain sense was disturbed first because the disease began as a meningitis and did not begin in the cord proper.

DISCUSSION

Dr. J. Hendrie Lloyd said that this was a case in which Dr. McIver and he had been very much interested. There had been some differences of opinion expressed in regard to the causation. The patient's appearance hardly supported the diagnosis of pernicious anemia. It was a secondary anemia not pernicious in type. But the patient had a double plus Wassermann; he had the symptoms of a posterolateral sclerosis, as had been described, and he had no pupillary involvement indicative of syphilis. The idea of pernicious anemia causing cord changes was of course not a new one. Dr. Lloyd had reported more than twenty years ago one of the best marked cases occurring in the Philadelphia Hospital. Recently, the subject has been much exploited. An author in the West said he has seen nearly three hundred cases of pernicious anemia in three years, which is certainly an extraordinary record. Dr. Lloyd said he himself had not seen three hundred cases in thirty years, or a number anywhere near that. In Minnesota they had the bothriocephalus latus, which caused an anemia very similar to pernicious anemia. The author in the West does not mention this; nor does he give a blood count or a Wassermann test, so Dr. Lloyd thought that altogether he had written a rather unsatisfactory and unscientific paper. The bothriocephalus tapeworm is a tapeworm that we do not have in this country unless it is imported. It prevailed more particularly in Scandinavian countries, and had been brought to Minnesota, where they had Scandinavian immigrants. Dr. Lloyd thought that some of those cases of so-called pernicious anemia in the West that were reported from Minnesota and Wisconsin were probably cases of Bothriocephalus anemia. Bothriocephalus anemia is like any other anemia of high grade in that it might possibly cause changes in the cord. Dr. McIver's case, however, had shown no symptoms of any tapeworm infection. Dr. Lloyd had once seen a case of Bothriocephalus infection, with a high grade of anemia, in a young Danish woman in Philadelphia. The anemia was of the pernicious type, and yet the patient had no marked symptoms of involvement of the spinal cord. In the present case he has not felt satisfied with the diagnosis of pernicious anemia, because the blood count and the appearance of the patient hardly supported it; and, on the other hand, the possibility of syphilis cannot be ignored. Moreover, a secondary anemia may be caused by syphilis.

Dr. William G. Spiller said that Dr. McIver brought up the subject as to whether syphilitic posterolateral sclerosis could produce the signs of a posterolateral sclerosis of anemic origin. He presumed it could if the involvement of the posterior columns was a meningitis with cellular infiltration into the posterior columns without involvement of the posterior roots, but this was not likely to occur.

The + + Wassermann reaction in Dr. McIver's patient was no proof that the changes in the spinal cord were syphilitic. A syphilitic man might have posterolateral sclerosis from anemia, and the findings in this case were typical of the anemic posterolateral sclerosis. The man had about 2,000,000 red blood cells. Dr. Spiller said he had had a case recently in which the hemoglobin was 71 per cent. and the red blood cells were 3,700,000; within two weeks the hemoglobin had fallen to 21 per cent., and the red blood cells to less than 2,000,000, and the man died.

Dr. F. X. Dercum regarded the case as in all probability one of anemia. It must be remembered, however, that in the lesions of the cord which gave rise to the syndrome of ataxic paraplegia there was an infiltration of the membranes of the posterior and lateral aspects of the cord and at the same time a marked interference with the lumen of the vessels, so that a degree of anemia was mechanically established in the portions of the cord affected. It was not improbable that this local interference with nutrition was the dominant factor in the production of the symptoms.

Dr. Charles K. Mills said that he had a private patient under observation in which this very question was more or less at issue. When Dr. Wilson and Dr. Mills first saw the patient three years ago, they made the diagnosis of an advancing posterolateral sclerosis of anemia. At that time the Wassermann test was negative. Dr. Mills did not see much of the patient until perhaps, two months ago, when he returned. He had since been under Dr. Mills' care, and had been seen somewhat frequently. His case had advanced very markedly when he returned; he was scarcely able to stand. Three years ago he had had increased reflexes, clonus and a Babinski reflex. Strange to say, now he has lost knee jerks and lost ankle jerks, and in that respect at least, the case had changed. Dr. Mills had had two Wassermann blood examinations made since his return; one of them had proved negative, and the other positive. It appeared to him that his own case was distinctly one of posterolateral sclerosis due to anemia, but the patient also may have had syphilis.

Dr. George Wilson said that he had carefully examined and questioned the man whom Dr. McIver had presented. He did not believe that the patient had real lancinating pains, which were so common in syphilis of the spinal cord. The man had told Dr. Wilson that the pains which he complained of were due to the involuntary jerkings of the lower extremities. Subjectively the patient had various paresthetic sensations in the hands and feet, a condition that was always found in the posterolateral sclerosis seen in anemia. There was no involvement of the bladder. The sensory loss was typically that described by Williamson and later by Dejerine in the combined sclerosis seen in anemia and other conditions such as pellagra. When syphilis produced a posterolateral sclerosis, pain sense was lost, diminished or retarded, exactly as in tabes, and the sensory loss which this man presented was not found.

Dr. Wilson said that the severity of the anemia in a given case had nothing to do with the possibility of that case developing cord changes. Three years ago Dr. Wilson examined in Dr. Mill's office a man with combined sclerosis of the anemic type who had 82 per cent. hemoglobin and 4,000,000 red cells. Careful examination of a blood smear revealed two nucleated red cells. A few months ago Dr. Wilson observed for some time a most extraordinary case of pernicious anemia in the service of Dr. A. A. Stevens in the Philadelphia General Hospital. That case had 10 per cent. hemoglobin and 225,000 red cells and yet presented no nervous findings, and today with a vastly improved blood picture showed no changes in reflexes, sensation or muscular power. The anemia did not produce the cord changes but rather the same toxemia which produced the anemia caused the combined sclerosis.

Dr. Wilson said that he believed that the + + Wassermann with the cholesterinized antigen was not worth considering, at least he had been so informed by serologists. Even though the patient had had a positive Wassermann reaction by the classical test, that could not change a diagnosis which to Dr. Wilson's mind was certain. The Wassermann test had led many astray; a great many cases with positive clinical evidences of syphilis were allowed to go untreated because the Wassermann reactions were negative and other cases with positive Wassermann reactions were not investigated further because they had a disease which might be present without being the cause of the syndrome found. As a case in point Dr. Wilson recalled a specimen of a spinal cord showing a tumor which Dr. Lloyd presented to the society. The Wassermann test was positive, but the tumor was beyond the syphilitic pale.

Dr. C. S. Potts said he thought, from what Dr. Lloyd had said, that his reason for not attributing the symptoms to anemia was that the blood did not show the picture of pernicious anemia. As Dr. Potts understood it, the condition could be caused by any form of anemia, and if he remembered correctly, a patient had been shown to this society several years ago in which the symptoms followed anemia caused by a severe hemorrhage. Chronic diarrhea and other exhausting conditions and toxemia have also been stated to be causes. The history and symptoms seemed in this case to be very characteristic of the so-called combined sclerosis which may follow pernicious and other forms of anemia.

Dr. J. H. W. Rhein said that he had a woman under his care at present who presented typical symptoms of posterolateral sclerosis, in whom the symptoms were practically identical with those seen in the case presented by Dr. McIver. The symptoms of the spinal involvement appeared when the blood picture was that of a secondary anemia. There were 3,700,000 red blood cells and 65 per cent. hemoglobin and an absence of anisocytosis and poikilocytosis. The color index was 1. In three weeks' time, however, the hemoglobin went to 60 per cent. and there were 1,910,000 red blood corpuscles present with some poikilocytosis and anisocytosis. Then under medication the blood picture became less like pernicious anemia, there being 3,720,000 red blood cells present and the hemoglobin advancing to 75 per cent.

He recalled a somewhat similar case which he had had under his care in the Polyclinic Hospital—similar to this one as far as the blood picture was concerned—and which presented the typical symptoms of posterolateral sclerosis without the typical findings in the blood of pernicious anemia. He commented on the fact that he had recently seen and heard of a few cases in which posterolateral sclerosis developed in anemias which were suggestive of, but not typical of, pernicious anemia.

It was possible, therefore, for the spinal cord changes to be quite far advanced before the blood itself showed the typical features of pernicious anemia, and while students of blood conditions will not admit that cases of this sort were really pernicious anemia, it was Dr. Rhein's belief that they belonged to the same group of blood diseases.

Dr. Alfred Gordon said that if we considered the general histories of syphilis of the cord, in the majority of cases in addition to the tract involvement there was involvement of the sphincters which was absent in the present case. He did not see why we hesitated to accept the possibility of anemia when the blood picture was typical of anemia; hemoglobin 60 per cent., and the red blood cells greatly decreased in number. The picture of anemia was clear. The presence of syphilis, as judged by the spinal fluid examination, was exceedingly doubtful. The posterolateral sclerosis of anemia was not such a rare thing. It had been reported, and personally Dr. Gordon did not feel any hesitation in accepting the diagnosis of posterolateral sclerosis due to anemia.

Dr. Joseph McIver said that he had always regarded the case as one of rather severe anemia. Whether or not it was a pernicious or secondary anemia, could only be settled by the blood picture. This had been done entirely to his own satisfaction by Dr. Rivas who had made the blood examination. He thought undoubtedly the case was one of posterolateral sclerosis such as is associated with anemia.

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PRACTICAL CONSIDERATIONS IN THE DIAGNOSIS OF PERIPHERAL NERVE INJURIES, WITH SPECIAL REFERENCE TO COMPENSATORY MOVEMENTS

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PHILADELPHIA

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I. INTRODUCTION

During the year of its active existence at Cape May, there passed through the U. S. Army General Hospital No. 11, at which the authors were stationed, 617 cases of peripheral nerve wounds. When the entire neurosurgical staff was transferred to Fox Hills, Staten Island, N. Y. (U. S. Army General Hospital No. 41), a large number of additional peripheral nerve cases were observed. These cases were studied with considerable care, and the findings fully recorded on forms especially prepared by the members of the Cape May neurosurgical staff. These forms were later adopted throughout the army for use in peripheral nerve hospitals. Shortly after admission each patient was examined by his ward surgeon and discussed at a conference of the neurosurgical staff. Re-examinations were made and the patients reviewed at conferences at intervals of from one to two months. In many instances patients have been operated on and the clinical diagnosis studied at this time in the light of the operative findings. By this procedure errors in diagnosis have been reduced to a minimum. The average case has remained under observation over three months so that ample opportunity for study has been afforded. The patients have all reached us some time after the occurrence of the wound, however, so that the observations which follow must not be taken to apply to early nerve wounds, but to injuries of over a month's standing.

As a result of these studies, some points have been noted which have not been sufficiently emphasized, or have escaped mention in descriptions of typical paralyses from nerve wounds. This paper will deal with this practical aspect of the subject rather than attempt a systematic discussion of nerve injuries, or the setting forth of debated theories.

II. FACTORS CONCERNED IN THE DIAGNOSIS OF PERIPHERAL NERVE LESIONS

A complete diagnosis should include the following points:

1. Identification of the nerve injured.
2. Description of the level at which the lesion occurs.
3. A statement of the degree of functional loss in the injured nerve.
4. Some note as to the status of recovery.
5. An estimate of the pathologic condition.

A. Slight Motor Function Distinguished from Complete Paralysis.—The first and second parts of the diagnosis generally present little difficulty. The fourth point is merely a matter of comparing the degree of function found at different times. The fifth (to determine the pathology existing in the nerve) is frequently impossible before operation. We are, therefore, particularly concerned with the third part of the diagnosis, for on this depends the whole question of treatment. It is nearly always possible to distinguish between complete loss of function, and the first faint traces of functional return. For correct observation on this point the patient looks to his surgeon to shield him from months of delay on the one hand, and unnecessary operation on the other.

B. Relative Value of Various Observations.—Although we have studied this problem from almost every angle, in the hope of finding some sure guide in the diagnosis of nerve injuries, experience has taught us to depend largely on the simpler diagnostic signs as being the most reliable. In the order of importance these may be rated: (1) voluntary movement; (2) sensation; (3) electrical reactions, and (4) trophic phenomena.

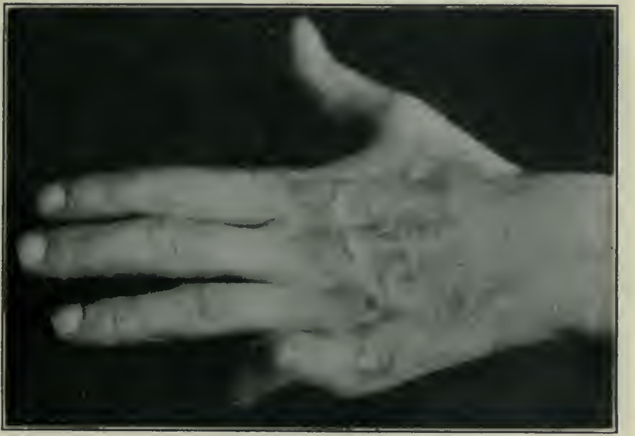

Fig. 1.—The typical hand of ulnar paralysis. The interosii are wasted, and the little and ring fingers flexed.

1. *Trophic Phenomena.*—First, it has been interesting to note that the trophic disturbances—the least important diagnostic sign—often enable one to make the diagnosis the instant the patient enters the room. So characteristic is the hypothenar and interosseous muscle atrophy in the ulnar cases, and the thenar atrophy in median injuries, that by examining plaster casts of the hands, one can pick out the median or ulnar cases nine times out of ten, provided brachial plexus lesions are excluded (Fig. 1). The trophic condition of the hand is, however, of very little value in showing the early signs of returning function. Indeed there may be the most pronounced degree of atrophy of the interosii, and yet all movements be executed with the intrinsic hand muscles apparently as well as though the hand were normal (Fig. 2).

2. *Electrical Reactions.*—Electrical examination has proved a disappointment to surgeons as it has failed to determine conclusively the degree of nerve injury or of nerve recovery. In two ways however, it is of real value. The faradic current generally settles quickly and definitely the differential diagnosis between a functional and an organic nerve lesion. If there is any question of the existence of a functional basis for a paralysis, and faradic stimulation gives a response in the supposedly paralyzed muscles, it is most unlikely that the paralysis is due to organic changes. Another important use of the electric current is in determining whether a muscle which now contracts, was once paralyzed. It has been observed that a recovering muscle often gives a slow, wavy response to galvanic stimulation, with a tendency toward a reversal of formula, even after it contracts voluntarily and responds to the faradic current. Therefore such a response as this, in a muscle which exhibits voluntary contraction, affords evidence that there was once paralysis. Attempts to determine the degree or nature of the injury by means of the electric current have, however, resulted in disappointment, since the familiar reactions of degeneration are frequently present in connection with spontaneous recovery, and may not be exhibited in their entirety in cases where the nerve has been completely severed.

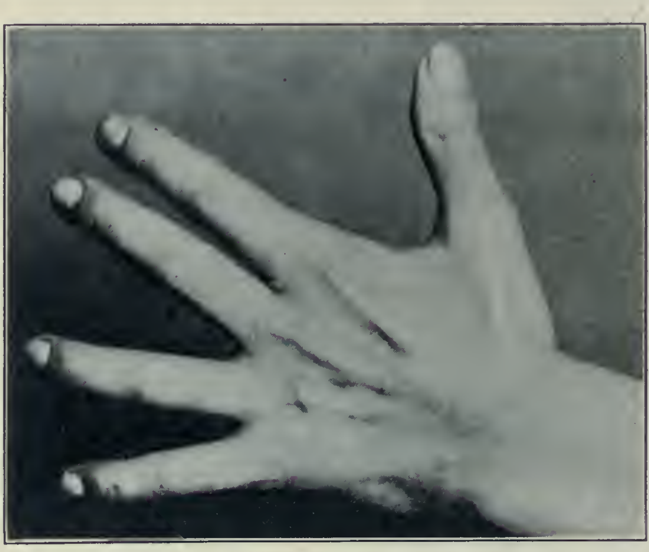

Fig. 2.—Ulnar atrophy in a case of ulnar paralysis in which function was returned in all ulnar muscles.

3. *Sensory Changes.*—The sensory examination is of considerable value, but unless each examination is made by the same observer, or with standardized instruments, the margin of error is apt to be wide.

In routine examinations for impairment of sensation, all but three sensory tests have been discarded. Pressure, pain and tactile sensation are tested, the esthesiometers devised by one of the authors (S. D. I.) being found the most satisfactory (Fig. 3). These esthesiometers were adopted as part of the neurologic equipment of the army peripheral nerve hospitals. Standard charts are used, and loss of deep pressure sense is shown by solid black. Tests for this loss are made by an esthesiometer which exerts a pressure of 1,000 gm. over an area of 0.25 sq. cm. Loss of pain sense is recorded by means of stippling within the anesthetic area which has been outlined on the chart. For this test an esthesiometer is used by means of which a pressure of 15 gm. is exerted on a sharp needle. Superficial tactile sensation is tested by means of a soft artist's brush, so trimmed that pressure over the most delicate portion of the skin does not cause visible depression of the epidermis. Loss of superficial tactile sensation is charted by means of hatching within the outline of the anesthetic area. Even when thus reduced to their simplest terms, sensory examinations require more time than all the other examinations combined; moreover, the sensory is of less value than the motor examination except in paralyses of purely sensory nerves, and in certain cases where returning sensation is the first evidence of recovery or regeneration.

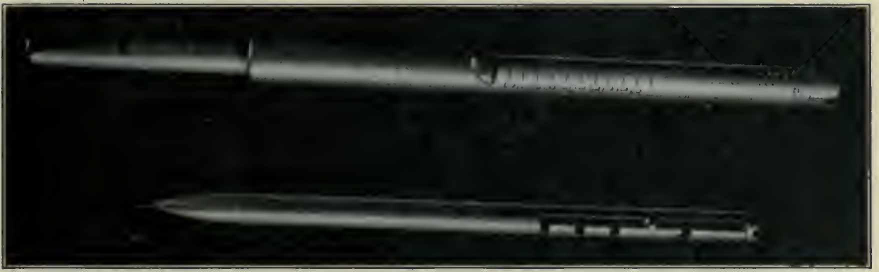

Fig. 3.—Esthesiometers devised by one of the authors, and used in the army peripheral nerve hospitals. The instrument above tests deep sensation, that below tests superficial pain sensation.

The test described by Tinel, consisting of distal tingling on percussion over a regenerating nerve trunk, has been found to be of confirmatory value.

Another phase of the sensory examination deserves mention, namely, spontaneous pain. When this pain is severe and burning in character and is increased by touching the skin over the painful area, it is known as causalgia. This symptom is considered as evidence of an irritative lesion involving the sympathetic nerves. The presence of causalgia, however, by no means precludes the possibility of an anatomic division of the nerve. In two of our cases causalgia was observed shortly after suture of the entire sciatic nerve.

4. *Motor Loss.*—Most important is the examination of motor function, just as motor paralysis constitutes the greatest part of the patient's disability from the standpoint of utility. Three observations are possible in testing the motor function of muscles: the effect of muscular contraction on the tendon, the effect on the joint and the effect on the muscle itself.

(*a*). Tendon Palpation.—Observing the effect of muscle contraction on the tendon by palpation is the most accurate method when it can be utilized. The observer may, however, be misled by the tightening of the tendon due to a change in the position of the joint, the resulting passive tension on the tendon being mistaken for the effect of muscle action. To eliminate such errors it is well so to fix the joint that no movement can occur, and then to have the patient attempt to move the joint. A tendon can in this way be made to stand out so as to be readily perceived, provided its muscle contracts. The factor of passive pull has been eliminated because the joint is immobilized.

(*b*). Observations of the Muscle.—We may dismiss with a few words the subject of observation on muscles. It is generally most unsatisfactory to attempt to see or feel the contraction over the belly of a muscle. Muscles in the vicinity, if they contract, will give an erroneous impression of action in the paralyzed muscle, while a slight true contraction will pass unnoticed. Exceptions to this rule are found in the muscles of the thenar and hypothenar groups, the supinator longus and the deltoid. Palpation and inspection of these muscles furnish very valuable evidence of muscle function.

(*c*) *Joint Action.*—The observation of joint movements is of great value in testing the function of muscles, but the interpretation of this observation is apt to be difficult, and erroneous conclusions may easily be drawn. For example, we might conclude that a patient necessarily had musculospiral function because he could extend the wrist. This is by no means the case. Probably all who have had an opportunity to watch patients with musculospiral lesions have seen many who obtain a certain degree of wrist extension, the musculospiral nerve being completely severed (Fig. 4). Extension is here produced by the action of muscles ordinarily not concerned in this movement. Actions of this type are generally designated as compensatory movements, and are particularly prone to occur in patients who have had paralysis for a long time, patients who have received special training in the use of their muscles, or patients in whom muscle contractures have occurred. Compensatory movements may be due either to the contraction of one set of muscles or to the relaxation of another. They may also be due either to the factors of gravity or momentum.

III. PRODUCTION OF COMPENSATORY MOVEMENTS

A. *Compensatory Movements Produced by Momentum.* — To understand the part which momentum plays in the production of compensatory movements, one may observe the patient with a fracture of the patella and separation of the fragments. He obtains extension of the knee by a swinging movement, thus substituting momentum of the leg for contraction of the quadriceps extensor muscle. The patient with musculospiral paralysis uses momentum in a similar manner, and frequently acquires considerable facility in extending the wrist by giving the hand a quick backward flip. For this reason joint action should not be taken as evidence of muscle contraction unless the movement is carefully analyzed by the observer, and the part so immobilized as to eliminate the momentum factor.

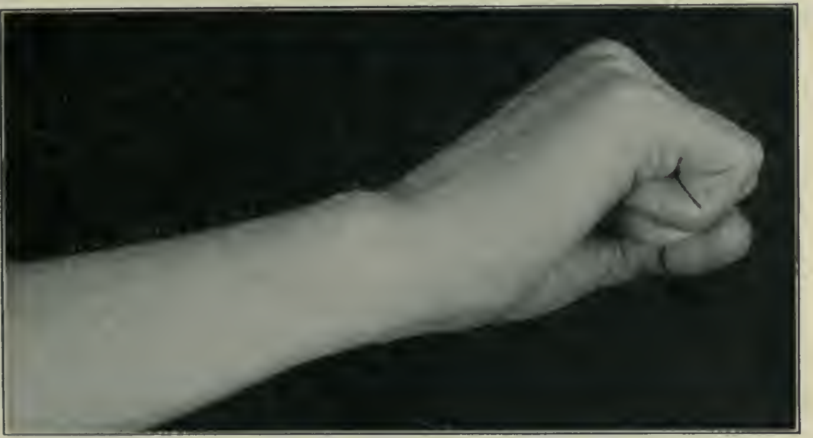

Fig. 4.—A case of complete musculospiral paralysis extending the wrist. This is accomplished by winding up the tendons of the extensor muscles (which have undergone contracture) by flexing the fingers.

B. *Gravity as a Factor.*—Gravity must also be considered as a means of producing compensatory movements. In musculospiral paralysis, the limp wrist which cannot be extended against gravity, falls into the position of extension if the hand is turned in supination. In order that the test may be perfectly reliable in showing all the motion that the patient is able to make, and at the same time rule out confusing movements due to gravity, the hand should be held midway between pronation and supination — the zero position. What is true of the wrist is also true of other joints; tests for muscle action should always be made with the joint in the zero position.

C. *Relaxation of Functioning Muscles.*—Far more confusing, however, than the movements produced by momentum or gravity, are those produced by other muscles which simulate the action of the paralyzed ones. Let us assume that we have a case of complete musculospiral paralysis with shortening in the extensor muscles, so that the hand naturally assumes the position of extension instead of wrist drop. If one were to grasp the hand and cause the wrist to flex, the hand would spring back into its extended position as soon as it was released. The patient can obtain the same result by the action of the flexors. These muscles pull the hand down, and when relaxation occurs, the hand again assumes the attitude of extension. When this movement is seen for the first time it gives a very misleading impression of extensor action, whereas the whole movement is really due to the flexors. Since the extension of the wrist is due to relaxation of the flexors instead of contraction of the extensors, it may be designated as a compensatory movement of relaxation. To eliminate this possible source of error, the flexors should be relaxed before the movement of extension is attempted. This is best accomplished by having the patient flex the wrist, slowly relax and then attempt slow, steady extension. No further movement of relaxation can occur under these conditions because the flexors are already completely relaxed. Any further movement of extension would therefore be evidence of musculospiral action, provided the relative position of the fingers and hand remains unchanged. The reason for this provision will be considered in the next paragraph.

D. *Contraction of Nonparalyzed Muscles.*—Let us again assume that we have a complete musculospiral paralysis, with contractures in the extensors of sufficient degree to prevent the hand from falling into the wrist drop position. If the examiner were to grasp the fingers and flex all of the phalanges until the hand assumed the position of a tightly closed fist, the extensor tendons, running over the summits of the knuckles, would be made more tense and would prevent the fingers from flexing unless some change in joint position could be secured which would lessen the tension. The wrist joint is the only one free to move which could release this tension. Extending the wrist joint tends to relax the extensors. This movement must therefore occur to allow the flexion of the fingers when the extensors have undergone contracture; and, indeed, this movement does occur when a patient with such contractures attempts to close the fist tightly, so that we have the curious spectacle of a man with musculospiral paralysis extending the wrist by flexing the fingers (Fig. 4). This type of movement may be characterized as a compensatory movement of contraction. On account of this movement of substitution, while testing for wrist extension as an evidence of musculospiral action, the relative position of the fingers and the hand must remain unchanged.

E. *Elimination of Compensatory Movements by Proper Methods of Examination.*—It would therefore appear, from a study of the various factors concerned in compensatory movements of the wrist, that: (1) The forearms should be immobilized to eliminate the factor of momentum, and the movement should be made slowly. (2) The hand should be in a position midway between pronation and supination to eliminate the factor of gravity. (3) Before testing extension, complete relaxation of the opposing muscles should be secured. This is best accomplished by having the patient first flex the wrist, then relax and finally attempt extension. (4) The relative position of the fingers and the hand should remain constant.

What is true of the wrist is also true of other joints, and the same precautionary measures should be taken in all cases to eliminate compensatory movements. In general, the part should be immobilized proximal to the joint, and the movement made slowly. A zero position should be secured to eliminate the factor of gravity, and complete relaxation should be secured in the opposing muscles. No movement in the joints distal to the one under examination should be allowed. Failure to observe these measures when a well marked compensatory movement exists will almost certainly lead, sooner or later, to errors in diagnosis.

IV. LESIONS OF INDIVIDUAL NERVES CONSIDERED IN REFERENCE TO COMPENSATORY MOVEMENTS AND OTHER PHENOMENA

The general principles governing compensatory movements have been discussed. These movements will now be studied in relation to the nerve lesions which have most commonly presented themselves.

A. *Circumflex Nerve Lesions.*—The circumflex nerve is frequently involved in shoulder injuries. In such cases an anesthetic area is found over a portion of the deltoid muscle, and generally the arm can be only slightly abducted from the side unless the scapula is rotated. Occasionally, however, a patient, without apparent contraction in the deltoid muscle, and with everything including sensory and electrical tests indicating a complete lesion of the circumflex nerve, is able to raise the arm to a horizontal position before the scapula commences to rotate, and considerably higher when aided by rotation of this bone. In the cases we studied at Cape May, this movement was due to supraspinatus action. The supraspinatus muscle, however, is not situated advantageously for producing abduction, and without the deltoid the shoulder usually cannot be abducted to any marked degree.

B. *Musculocutaneous Nerve Lesions.*—Musculocutaneous paralysis results in loss of sensation over a portion of the forearm, and weakness in supination and elbow flexion. Although the brachialis anticus and biceps are completely paralyzed, there is not complete loss of any movement of the elbow because the supinator longus, a powerful flexor of the elbow which is innervated by the musculospiral, partially substitutes for the muscles supplied by the musculocutaneous when it is paralyzed. When both the musculocutaneous and the musculospiral are injured, one would expect to find flexion of the elbow and supination abolished. This is often the case. Supination was abolished in all of our cases, but in a few instances flexion of the elbow was obtained through a compensatory movement of contrac-

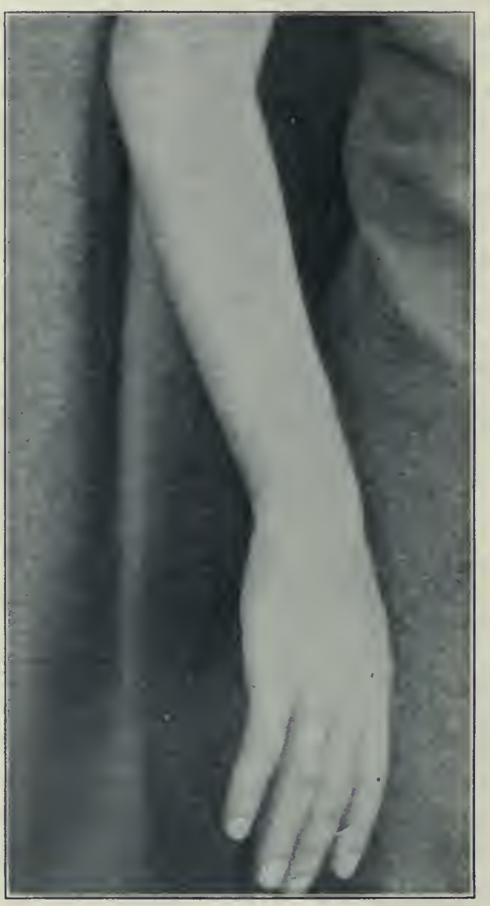

Fig. 5.—Patient with musculospiral and musculocutaneous paralysis with muscles relaxed.

tion by the pronator radii teres, which is supplied by the median nerve. This muscle, having its origin at the internal condyle of the humerus, and its insertion in the shaft of the radius, is so situated that it does not act with sufficient mechanical advantage to produce strong flexion; but when a patient has trained his power of flexion by exercise, a sufficiently strong contraction may be obtained to flex the elbow to 90 degrees (Fig. 5), and to lead the patient (and perhaps also the physician) to believe that function is returning in the injured nerves. Palpation over the supinator longus and biceps and all other tests at our disposal will, however, show these muscles to be completely paralyzed, whereas the pronator radii teres will be found to contract strongly while the elbow is being flexed. Pronation accompanies the movement. Thus, we find that the musculospiral nerve always partially compensates for the loss of function of the musculocutaneous, and that even when both of these muscles are paralyzed slight compensation is occasionally secured through the pronator radii teres.

C. *Musculospiral Nerve Lesions.*—Loss of musculospiral function results in inability to extend the elbow, wrist and thumb. The metacarpophalangeal joints of the fingers cannot be extended. Besides this there is found, as in the case of musculocutaneous paralysis, weakness of supination and elbow flexion, without complete loss of either, the biceps supplementing the supinator longus and brevis in these movements. It is sometimes difficult to tell whether action in the supinator longus is absent or merely weak, since contraction of the biceps tends to pull on the fascia of the antecubital space, giving an erroneous impression of muscle contraction in the supinator longus. In such cases it is well to fix the forearm by holding the wrist, and then to have the patient attempt to flex the elbow while the palpating finger is applied on the supinator longus, over the shaft of the radius, several centimeters below the insertion of the biceps tendon. At this point any contraction of the supinator longus may readily be discerned.

It is to be noted that in musculospiral paralysis, extension of the interphalangeal joints of the fingers is possible, being accomplished by the interosii and lumbricales (Fig. 6). This movement in some cases is so marked as to simulate musculospiral action. Errors of interpretation from this source may be avoided by carefully observing the metacarpophalangeal joints. No true extension will be found if the case is a complete musculospiral injury.

Low lesions of the musculospiral may be overlooked because of their failure to produce wrist drop. Extension of the wrist is secured by the action of the extensor carpi radialis longior. The branch from the musculospiral to this muscle comes off well above the elbow; therefore a lesion below this point cannot produce wrist drop.

Compensatory movements in musculospiral paralysis in their bearing on the wrist joint have already been discussed, and nothing further need be added. If the examiner is on the alert for these movements and takes the precautions which have been suggested to eliminate compensatory movements of contraction and relaxation, and the factors of gravity and momentum, he should have little difficulty in securing an accurate estimate of the degree of musculospiral loss.

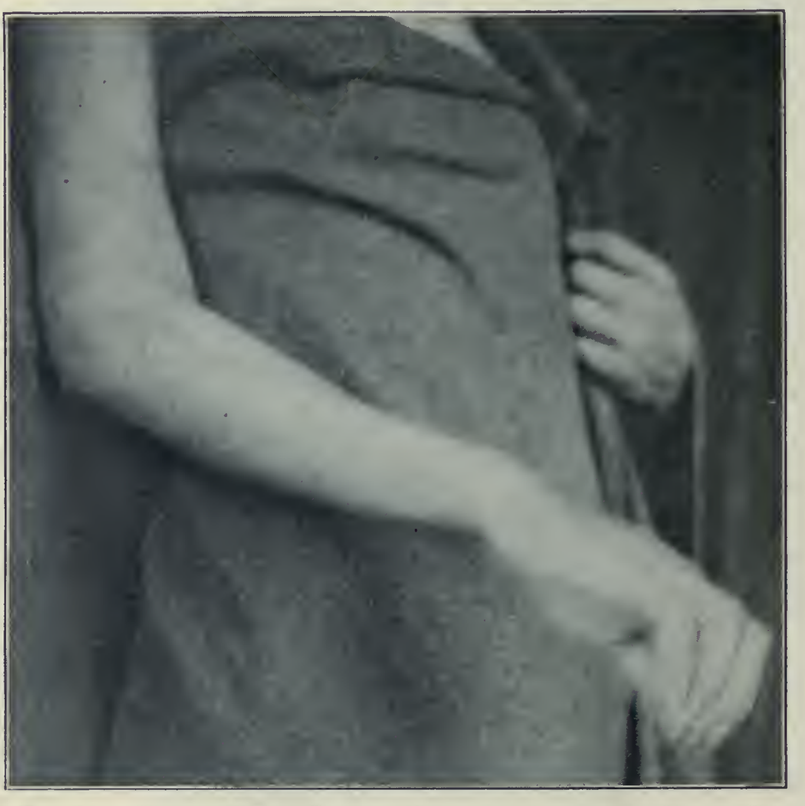

Fig. 6.—Same patient with flexion of the elbow obtained by action of the pronator radii teres.

D. *Median Nerve Lesions.*—Loss of function in the median nerve, besides resulting in loss of sensation over a portion of the hand and fingers, produces loss of thumb flexion and thumb abduction. Next in importance is the marked weakness in flexion of the index finger; there is also weakness in pronation, wrist flexion, apposition of the thumb against any other finger and in flexion of the fingers. No commonly seen peripheral nerve injury affords greater difficulty in diagnosis than does the median, partly on account of the tendency of the musculospiral and ulnar sensory functions to overlap the median, but particularly on account of the large number of compensatory movements that patients with median paralyses display. When the median nerve is severed, pronation may be begun by the action of the supinator longus and finished by the action of gravity so that an almost normal range of movement is secured without the action of the pronator radii teres and pronator quadratus muscles. Wrist flexion is likewise secured by the synergic action of the flexor carpi ulnaris, extensor ossis metacarpi pollicis and that portion of the flexor profundus digitorum which is supplied by the ulnar nerve. Because of this partial ulnar supply to the flexor profundus digitorum, flexion of the fingers is not lost in median lesions.

Flexion of the thumb may occur through a mechanism of relaxation to which we have already referred, this movement being dependent on the pull of the shortened flexor longus pollicis in the relaxation period following the action of the extensors. Flexion may also be secured by wrist extension when the flexors are shortened. This is another example of a compensatory movement of contraction. Apposition of the thumb to the fingers may be secured by the synergic action of the adductor pollicis, the deep head of the flexor brevis pollicis and the extensors of the thumb.

Abduction of the thumb may be secured in the absence of the abductor pollicis by the combined action of the deep head of the flexor brevis pollicis, which is supplied by the ulnar nerve, and the extensors of the thumb. This movement of substitution is not common, and is generally incomplete, so that loss of thumb abduction is one of the best signs of the absence of median motor function in the hand. A convenient and practical test for this function is to have the patient lay the hand, back down, on a table and attempt to point the thumb straight upward; or the examiner may place the patient's thumb in this position. Inability on the part of the patient to maintain this position of the thumb is evidence of loss of median action.

E. *Ulnar Nerve Lesions.*—Ulnar lesions produce the well-known anesthetic area with a fair degree of constancy. This furnishes an important diagnostic sign, and constitutes a leading factor in the production of the disability which results from such injuries. Destruction of the ulnar nerve results in some cases in surprisingly little motor loss which cannot be compensated for by muscles supplied by other nerves. Generally, the only motion completely absent is abduction and adduction of the fingers.

Flexion of the fingers, with the exception of the thumb, is provided for by the combined action of the median and ulnar nerves. Finger flexion is present, though perhaps weak, in paralysis involving either one or the other of these nerves. Bearing in mind that the portion of the flexor profundus digitorum, which has its insertion into the little and ring fingers, is supplied by the ulnar nerve alone, one would expect that in ulnar paralysis the terminal phalanges of the little and ring fingers could not be flexed. This is not the case as a rule, because apparently all of the tendons of the deep flexor muscle are acted on to some degree when either the median or the ulnar portion contracts; hence the little and ring fingers are usually flexed in their terminal portions when the index and middle fingers flex. A fairly satisfactory test for function in the ulnar portion of the flexor profundus digitorum is obtained as follows: Completely immobilize the index and middle fingers, and immobilize the little and ring fingers up to the terminal phalanx; then have the patient attempt flexion of the fingers. In the absence of ulnar function, the little and ring fingers cannot be flexed in their terminal phalanges.

In ulnar paralysis the loss of the flexor carpi ulnaris may be compensated for by the action of the extensor carpi ulnaris. This muscle pulls the hand toward the ulnar side, while flexion of the entire wrist is attained through the action of the median flexors of the wrist.

Loss of the lateral movements of the fingers is probably the most distinctive sign of ulnar paralysis. Strangely enough, however, the extensors may be trained to produce lateral movements of the fingers, probably because the extensor tendons do not usually run directly across the apices of the knuckles, or if they do, they are inserted laterally into the phalanges. The action of the lumbricales also tends to produce lateral movement of the fingers. The lateral action which is sometimes found in the little and ring fingers in ulnar paralysis cannot, however, be explained in this manner. A useful method for overcoming this compensatory movement is to put the extensor tendons under tension by flexing the wrist and fingers before attempting the movement of spreading the fingers. Another useful procedure is to palpate the hypothenar eminence while the little finger is abducted. The abductor minimi digiti contracts if ulnar action is present to any marked degree, and the skin over the hypothenar eminence wrinkles and dimples inward as the muscle contracts. The latter action is due to the contraction of the palmaris brevis which is supplied by the ulnar nerve and inserts into the palmar fascia.

F. *Ulnar Combined with Median Nerve Lesions.*—When ulnar and median lesions coexist, flexion of the wrist and fingers is no longer possible except when compensation is secured by means of the factors already discussed. The extensor ossis metacarpi pollicis furnishes the most important action in this respect; a surprising degree of flexion is often obtained by contraction of this muscle (Fig. 7).

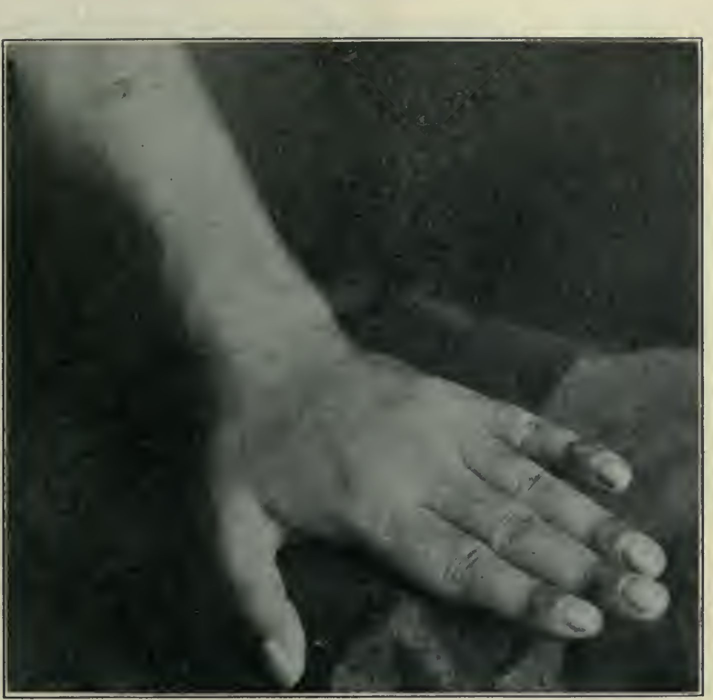

Fig. 7.—A patient with complete musculospiral paralysis extending the interphalangeal joints by means of the interosii and lumbricales.

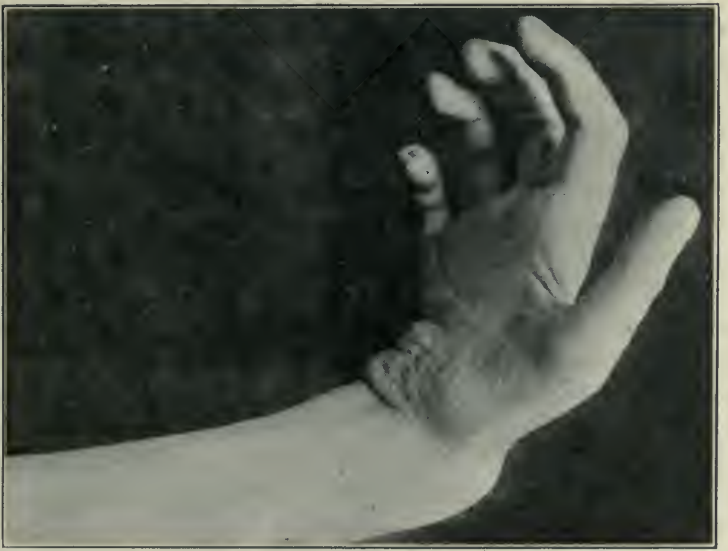

Fig. 8.—Flexion of the wrist by means of the extensor ossis metacarpi pollicis. The case is one of complete ulnar and median paralysis.

G. *Sciatic Nerve Lesions.*—Lesions of the sciatic nerve when complete usually cause loss of all movements below the knee. A striking fact in this connection is that even when the lesion is found to be high on the sciatic nerve, frequently no paralysis of the hamstrings occurs. Another peculiarty of sciatic lesions is the tendencey toward dissociation which they display. Lesions high in the thigh will often produce paralysis only in the internal or external popliteal supply, usually in the latter.

H. *External Popliteal Nerve Lesions.*—In lesions of the external popliteal nerve one finds loss of sensation over the lateral aspect of the foot and leg, inability to evert or extend the foot, and loss of extension in the metacarpophalangeal joints. As in the hand, the distal phalanges can be extended even in the presence of a paralysis of the extensor muscle, the extension being accomplished through the agency of the interosii and lumbricales. All of the types of movements of compensation found in the upper extremity may occur, and this source of error may be overcome by the means already described. Because movements of the toe and ankle joints tend to be more restricted than finger and wrist movements, tendon palpation is a more valuable guide to muscle contraction in the lower extremity than joint action. Before the toes or ankle commence to extend, it is often possible to feel the extensor tendons become taut where they run beneath the anterior annular ligaments of the ankle.

I. *Internal Popliteal Nerve Lesions.*—Internal popliteal lesions are accompanied by anesthesia on the sole of the foot. The motor disturbance consists in loss of plantar flexion of the foot and toes, absence of lateral movements of the toes and inability to evert the foot. A fairly common and somewhat confusing compensatory movement may occur. Some patients obtain plantar flexion of the foot by use of the peronei, which are supplied by the external popliteal nerve. When these muscles contract even when the internal popliteal nerve is completely paralyzed, there may result a diagonal tilting of the foot which may be mistaken for plantar flexion; true plantar flexion may even occur in cases where the peronei insert well back of the axis of rotation of the ankle. Other types of compensatory movements are found to some degree in connection with internal popliteal lesions, but if proper precautions are taken none will be likely to lead to an error in diagnosis with the exception of that to which we have just alluded. This confusing compensatory movement should be suspected if, in the presence of other evidences of internal popliteal paralysis, the patient obtains feeble plantar flexion of the foot without evidence of gastrocnemius contraction. In such cases palpation of the peronei will demonstrate contraction of these muscles, but the Achilles' tendon will be found to relax instead of tighten with each downward movement of the foot. When these conditions exist, whatever plantar flexion is obtained is the result of a compensatory movement of contraction by the muscles supplied by the external popliteal nerve.

V. CONCLUSION

The list of compensatory and substituted movements might be prolonged indefinitely, but enough have been considered to emphasize the importance of careful observation to avoid errors in diagnosis. In examining the results of a large number of peripheral nerve lesions, the characteristic and classic symptoms will commonly be found; however, a certain number of cases will exhibit unusual phenomena. These atypical cases are the ones that offer the greatest difficulties in neurosurgical diagnosis.

It is hoped that sufficient has been said to show the inadequacy of simply learning a list of the classic symptoms as signs of nerve injuries. The fundamental requisites for accurate diagnosis of such cases include a thorough anatomic knowledge, a mastery of the mechanics of joint action and discriminating observation. With the application of these broad principles to neurosurgical diagnosis, the difficulties are minimized and the proper treatment can be confidently instituted.

A CONSIDERATION OF THE NATURE OF AURAE *

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Descriptive and statistical studies of aurae are to be found in textbooks of nervous and mental disease, treatises on epilepsy and migraine and special articles; but these sources of information are searched almost in vain for an exposition of the particular conditions of the nervous system or of the mind that favor the occurrence of aurae. Such references as one finds generally are bare statements unsupported by argument and obviously taken without question from the conclusions of some distinguished investigator. That these references are scarce and unconvincing is not surprising in view of the difficulty of investigating aurae, as well as other phenomena connected with epilepsy and migraine, first hand in the person afflicted.

The subject would seem to be worthy of consideration, however, because without a proper understanding of the underlying conditions it is hardly possible either to form a satisfactory opinion of the relation of aurae to the convulsions in epilepsy, or to the headaches in migraine; or to make an accurate statistical or analytic study of aurae themselves. In this article, therefore, an attempt is made first, to bring forward objections to what seems to be the prevailing view; second, to point out anew the relation of aurae to a certain mental state, and finally, to suggest some factors that may influence the form the aurae may take.

AURAE OF EPILEPSY

Objections to Prevailing View on Aurae.—The notion of aurae that is most popular with the authors of textbooks probably originated with Hughlings Jackson,[1] but is given very clearly and concisely in these words of Gowers:[2]

"This immediate warning is of the greatest importance . . . The sensation is the effect on consciousness of the commencing process of discharge, which begins in structures by which these are influenced that are highest in function and related closely to consciousness. The warning is thus an indication of the part of the brain in which discharge begins, because the place where it first attains such an intensity as to cause a sensation, must be the place at which it starts. . . . Their interest is great, but their practical value is almost limited to the detection of attacks which might otherwise be unperceived, by the evidence their character affords, and by their frequent indication that the instability is the result of a local lesion of the brain." There is in this conception of the nature of aurae an application to the sensory component of the epileptic attack of one theory of the origin of convulsions, namely, that which assumes them to be the result of a "discharge" of nervous energy in the motor cells of the precentral area.

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* Contribution in a series offered to Prof. E. E. Southard in honor of the decennium of the Bullard Professorship of Neuropathology, Harvard Medical School.

[1] Jackson, H.: Brain **11**:179, 1888.

[2] Gowers: See Albutt-Rolleston: A System of Medicine, Ed. 2, New York, Macmillan Co. **8**:494.

Even though this conception of the origin of the convulsive movements of epilepsy be accepted as satisfactory, its application in the explanation of aurae may be objected to. The convulsive movements are of sudden onset and display little evidence of a coordination of nervous processes. On the other hand, the aura may be relatively slow in evolution and if complex in nature, indicates a delicate coordination of nervous impulses that is inconsistent with the notion of a "discharge." Further, the conception is incomplete in that it does not indicate the relation of the aurae which occur in the presence of an organic lesion to those which occur in brains apparently normal in structure. It is begging the question to get over this difficulty by assuming all cases of epilepsy to be the result of an organic brain disease. Structural changes that probably are not secondary are not found in a large proportion of the brains of a certain type of epileptics. In a still larger proportion the aura is not associated with changes in special sense centers that correspond to the special sense in which the aura occurs. It cannot reasonably be stated, then, that the aura of a special sense indicates or may indicate structural or even functional changes of a pathologic nature in the centers of that sense. A theory of aurae should make clear in how far a particular aura may be determined by structural changes and where these changes should be looked for.

In view of the relative futility of efforts to investigate aurae first hand in afflicted persons, it is well to attempt to employ another method, i. e., try to get some light from a study of similar and perhaps less inaccessible phenomena, if such can be found, and to apply the information thus obtained by analogy. There is a mental phenomenon, or rather a series of allied phenomena, of common occurrence in man, namely, hallucinations, to which it would seem aurae could be safely compared. Parish,[3] McDougal[4] and others have noted the similarity of these manifestations of mental activity. Although some conspicuous differences may immediately stand forth, it is apparent that they are not essentially dissimilar. The subject matter of the one can generally be essentially duplicated in the other. The senses of sight and hearing, moreover, are represented in both in a similar proportion — as 71 to 29 in some statistics of the occurrence of aurae collected from several sources, and as 70 to 30 in the statistics of the occurrence of hallucinations, collected by Parish.[5] The half-recalled memories or "deja vu" phenomena of certain incomplete attacks have many parallels, occurring in connection with sleep and certain waking mental states. No insuperable objection to this comparison is evident, at first sight at least.

Relation of Aurae to Other Hallucinations.—Hallucinations are common in both abnormal and physiologic mental states. Their occurrence in the insane and in those addicted to the use of drugs and alcohol is well known. The most common manifestation in physiologic states occurs in connection with sleep, in the form of dreams. There are also the hallucinations of hypnosis, of crystal gazing and of the psychic medium. Finally, there is the spontaneously occurring variety so much studied by spiritualists, and commonly known as visions.

According to Parish,[6] there is a common mental state underlying all hallucinations, which he designates "dissociation of consciousness." By this term he refers to a state of consciousness that differs from the waking state, in that normal association of ideas does not occur, or in which, as James expresses it, ideas are "deprived of their reductives." Leaving aside as too complicated for consideration the pathologic states in which hallucinations are found, there is the loss of consciousness to a greater or less degree in sleep and a similar, but less profound, loss in hypnosis and crystal gazing, which favors the production of hallucinations. That the psychic medium usually gives her information with eyes closed, would indicate that she may be at such times in a state of autohypnosis. In the case reported by Prince,[7] visions appeared in a prospective medium only after she had gone through a preliminary course of training, which consisted in gazing intently at a small point on a table for a long time. She doubtless was thus acquiring the power of autohypnosis. In the case of the visions reported by various societies for psychical research, Parish was able to demonstrate that in a large proportion they occurred under conditions that favor sleep, or during emotions such as grief, fear or excitement that may produce a somewhat similar dissociation of consciousness. Before reading the conclusions of Parish, I had been impressed with this association merely in going over accounts of visions by various authors.

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[3] Parish: Hallucinations and Illusions, New York, 1909.

[4] McDougal: Hallucinations, Encyclopedia Britannica.

[5] Parish: Hallucinations and Illusions, New York, 1909, p. 107.

[6] Parish: Hallucinations and Illusions, New York, p. 71.

[7] Prince: Brain **21**:528, 1898.

There is in epilepsy a disturbance of consciousness in the form of a loss. The depth of the unconscious state precludes the recollection of any visions occurring at this time; but there is a brief interval before consciousness is entirely lost that is probably not unlike the normal drowsy state and during which there are probably the conditions which have been shown to be suitable for the development of hallucinations. That hallucinations or aurae do occur at this time is, therefore, not surprising. Hence one should consider aurae as having an origin somewhat similar to that of hallucinations in normal persons, and should not assume, as Gowers and others have assumed, that they result from a "discharge" of an epileptic nature, due perhaps to a local lesion.

Factors Influencing the Form of Aurae.—If this assumption be true, the content of aurae should in general be derived as are dreams and visions. They may represent a fragment of memory, as was the case with one patient of mine whose aura consisted of a familiar face —not always the same face, however—and with another, whose aura was the vision of a disagreeable old woman he had known in childhood. In this connection it is interesting to recall the suggestion of Schwab[8] that aurae may represent memory pictures of events attending the first fit, which have been "fixed," so to speak, by the strong attending emotional reaction. Further, the aura may consist of diminution or distortion of normal perceptions — micropsia, macropsia, etc. — which occurs also in connection with sleep. Finally, the underlying factor may be structural change. Since disease of the organs of special sense or of their pathways leading to the brain may lead to the formation of hallucinations in the corresponding sensory spheres in either the sane or the insane,[9] similar changes may influence aurae. Turner[10] considered these factors, in the case of auditory aurae, without much success; but the subject is worthy of further study.

Some points at which aurae and other varieties of hallucinations differ are worthy of consideration. There is, first, variability; aurae being constant in most cases and hallucinations generally fleeting. This difference may be in part explained by one of the characteristics of epilepsy, namely, stereotypy. In general, the various reactions in epilepsy tend to be constant: hence, the preconvulsional disturbance of consciousness should be about the same in all attacks. This being true, the aura would be expected to be relatively constant. On the other hand, drowsy states vary within the widest limits and in them, unlike epileptic mental states, consciousness is often very receptive to external stimuli. It is therefore not surprising that the hallucinations should vary. Another point of difference is the absence of hallucinations during and after the convulsion. The depth of unconsciousness and fatigue factors may account for this lack. Some authors have assumed, however, that hallucinations do occur at this time.

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[8] Schwab: Unpublished contribution presented before St. Louis Neurological Society, 1916.

[9] Colman: Brit. M. J. 1894, p. 1015.

[10] Turner: Epilepsy, Chicago, Chicago Medical Book Co., 1907, p. 81.

AURAE OF MIGRAINE

As regards migraine, the problem is more obscure. Parallels for some of the aurae of migraine, such as the fortification spectrum described by Gowers for instance, are rare; but as aurae of this type seldom appear, few parallels should be expected. In visions and hallucinations bright lights and brilliantly colored objects are not unusual. Not unlike some migrainous aurae, is the vision of the boy, described by Gurney,[11] who saw in the dark a distant, tiny, luminous point approaching and increasing in size and becoming a face, and finally a mass of luminous faces unlike anything he had ever seen. A patient of mine had, after retiring and before falling asleep, a vision consisting of a "head of flame." That some disturbance of consciousness occurs with migraine there can be little doubt. Migraine is regarded by many as akin to epilepsy, and various abnormal sleep-states are commonly associated with it. One would think that conditions in migraine are even more favorable for the formation of hallucination than are those in epilepsy, inasmuch as in migraine the march of symptoms is slow and consciousness is not lost.

There remains to be considered the relation of the localized brain lesion to aurae. That there is an association of olfactory aurae with certain local lesions Hughlings Jackson,[12] Gowers[13] and Southard[14] have conclusively demonstrated. This fact instead of controverting the view that aurae are merely a type of hallucination, is itself made clear by some characteristics of hallucinations. It already has been pointed out that lesions of organs of special sense or of their pathways leading to the brain, may predispose to aura formation. In the case of the brain lesion, then, it may be assumed that the same principle is at work, the only difference being that interruption of peripheral impulses occurs in the brain rather than in the peripheral pathways. Indeed, this principle can be applied in instances where another, let us say, that of direct irritation to the cortical cells, cannot be. In a case with olfactory aurae described by Jackson,[12] for instance, the lesion found postmortem was a cyst of softening in the hippocampal gyrus. According to a dictum of Jackson himself, a purely destructive lesion should not be considered as acting as an irritant on surrounding cortical substance. Again, in a case with a similar type of aura, quoted by Gowers,[13] the lesion found at necropsy was a gumma of one olfactory lobe. In neither of these cases, then, can the aura have resulted from an irritation of the olfactory cortex; but both are easily explained as the result in the center of a disturbance of incoming nervous impulses.

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[11] Gurney: Phantasms of the Living **1**:400, 1886.

[12] Jackson: Brain **21**:580, 1898; Jackson and Stewart: Ibid. **22**:534, 1899.

[13] Gowers: Brain **32**:303, 1909.

[14] Southard: Am. J. Insan. **64**:607, 1907-1908.

CONCLUSION

By way of summary, it may be stated that I have attempted to point out anew the analogy between aurae and the hallucinations occurring in connection with sleep, hypnosis, crystal gazing, etc. According to this view, aurae should be regarded not as the result of "discharges" of an epileptic nature in some part of the cortex, but as deficiency reactions, like dreams, occurring when there is a "disturbance of consciousness" of a certain type. Their relation to the loss or disturbance of consciousness in epilepsy and migraine is assumed to be the same as that of dreams to drowsy or sleep states; and their content should be regarded as being determined by the same factors that determine the content of dreams and similar hallucinations. Their relation to structural changes may be the same as that of those hallucinations which develop in connection with disease of the organs of special sense or of the nerves connecting them with the brain.

A CONTRIBUTION TO THE HISTOPATHOLOGY AND HISTOGENESIS OF SYRINGOMYELIA *

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There still reigns a great confusion of opinions relative to the nature and origin of spinal cord cavities in general, and of those known as syringomyelia in particular. Thus, Schlesinger,[1] after extremely detailed discussion (covering 140 pages) concerning the pathology and pathogenesis of syringomyelia, comes to the conclusion that it may be caused by many anatomically different processes, and offers the following anatomic classification of spinal cord cavities:

1. Cyst formations (caused by injuries of the spinal cord, i. e., traumatic, also nontraumatic hematomyelias).
2. Softenings of inflammatory and noninflammatory origin, with a short course of the morbid process.
3. "Syringomyelien," in the form of: (*a*) malformation; (*b*) an actual tumor formation; (*c*) syringomyelia gliosa; (*d*) syringomyelia from vascular disorders without gliosis, and (*e*) pachymeningitis and leptomeningitis combined with cavity formations.

Schlesinger thus accepts at least five types of syringomyelia, each type differing from the rest anatomically and pathologically. If such be the case, syringomyelia cannot be considered a specific morbid condition, and clinically should be looked on rather as a syndrome. Such a view is shared by practically the greatest majority of students of this disease. For instance, Thomas and Quercy[2] claim that syringomyelia is devoid of any specific anatomopathologic features, and that it would be more proper to substitute the name of medullary cavities for that of syringomyelia, as, in their opinion, there are "syringomyelias" caused by gliomas, developmental anomalies, traumas, hematomyelias, softening, etc.

On the other hand, Phillippe and Oberthür[3] would call the latter types rather pseudosyringomyelias, referring to syringomyelia proper

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* From the pathology laboratories of Cook County and Psychopathic Hospitals of Chicago.

[1] Schlesinger, H.: Die Syringomyelie, Ed. 2, Wien und Leipzig, 1902.

[2] Thomas and Quercy: Syringomyélie, hyperplasie du tissue conjonctif, fibres musculaires striées dans la moelle, Nouv. iconog. de la Salpêtrière **25:** 364, 1913.

[3] Phillippe, C., and Oberthür, J.: Contributions à l'étude de syringomyélie et des autres affections cavitaires de la moelle épinière, Arch de méd. expér. et d'anat. path. **12:**513, 1900.

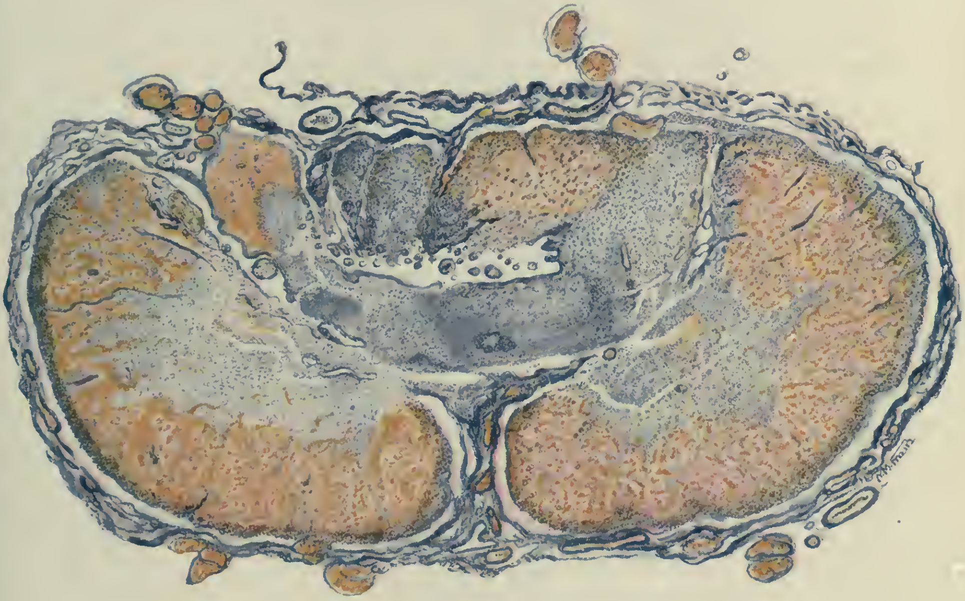

Fig. 9.—Syringomyelia; cervical enlargement. The pia, myelin, axons, gray matter, the pathologic glia, the marginal glia stained in different colors. Combined Bielschowsky-Alzheimer-Mann stain.

Illustrating Article by G. B. Hassin, M.D.,
Archives of Neurology and Psychiatry, February, 1920

only those morbid conditions that possess a certain specific pathology which they claim cannot be confounded with any other morbid condition. Though admitting two types of syringomyelia, cavitary and pachymeningitic, Phillippe and Oberthür believe that they are independent of each other, though caused by the same as yet unknown etiologic factor. The foregoing references represent prevailing opinions relative to the nature of syringomyelia. In spite of an enormous literature bearing on the subject, I believe my findings are of sufficient interest to be recorded.

From the study of my case, as well as from the most important previous contributions to this question, the conclusion may be drawn that in syringomyelia we do possess a number of specific pathologic changes which totally differ from those to be found in any other spinal cord lesion—changes which stamp syringomyelia as a distinct anatomopathologic entity.

REPORT OF CASE

History.—A white, married laborer, aged 54, entered Cook County Hospital, April 26, 1919, on the dermatologic service of Drs. Harris and Stillians, complaining of "sores" on the right arm and left shoulder. About three weeks previously his right arm felt lame, and showed a small, painless, red lump. Two weeks later this lump broke, discharging very foul-smelling pus. The sore on the left shoulder was also painless and of but two days' duration.

He had been a patient at the county hospital thirty-four years before for some trouble with the right eye, which was operated on, but he did not know the nature of the trouble. He had chancre twenty-five years before; and gonorrhea, twenty and two years before. He used to drink. His family history was of no importance.

Examination.—The patient was rather poorly nourished without any deformities, except an opacity over the right pupillary area and scars from an old iridectomy. On the right shoulder there was an ulcer, about 1½ inches in diameter, roughly circular, with a clear-cut margin; the edges were not thickened or everted, exposing the muscles beneath, which formed the floor of the ulcer; there was a scanty, yellowish discharge. The surrounding skin was reddened, scaling and, like the ulcer, painless. Lower down on the same arm, over the deltoid insertion, as well as over the left shoulder, similar ulcers were developing. The rest of the skin, on the back and lower limbs, was covered with "bites," scratch-marks and scars. Over the right hip was a discolored area. The muscles of the arms and both shoulders were atrophied.

Sensibility.—The temperature was abnormal. The pain sense was markedly impaired, the touch less so. The patient was rather indifferent to examination and cooperated poorly; yet it could be demonstrated that the entire surface of the body, including the upper and lower limbs, but excluding the face, was anesthetic, the anesthesia comprising the pain, temperature and touch senses.

Reflexes.—The pupillary (on the left) was sluggish; cremasteric and abdominal, normal; tendon reflexes, markedly exaggerated, with bilateral clonus, positive Babinski, Oppenheim, Gordon and Schaffer. The gait was uncertain, ataxic; the muscle power, greatly diminished. There were no tremors. The serologic examination gave a negative globulin, and a negative Wassermann test in both the blood and the spinal fluid, 20 cells per cubic milimeter and a negative Lange test (0011210000).

Course.—The ulcers grew larger, spreading over the scapulae, exposing the heads of the humeral bones and copiously discharging pus. Involuntaries (bladder and rectal) set in; the patient became totally paralyzed, apathetic, filthy. The tendon reflexes disappeared entirely; the sensory disorders became more marked, and two months after his entrance into the hospital he died.

Macroscopic Examination of the Spinal Cord and Meninges.—The dura was of normal thickness with smooth, shiny surfaces, without adhesions, pseudomembranes, hemorrhages, or any other pathologic deposits. The pia appeared thickened, especially around the cervicodorsal region and adherent to the spinal cord. The latter was greatly flattened in the mid-dorsal region. In the cervical region, which was somewhat enlarged, there was a small cavity, surrounded by a gray area occupying both posterior horns and the greater part of the posterior columns. The upper half of the dorsal portion was very friable, granular in appearance and surrounded by remnants of white matter. The lumbosacral segments were of normal configuration, but on section exhibited

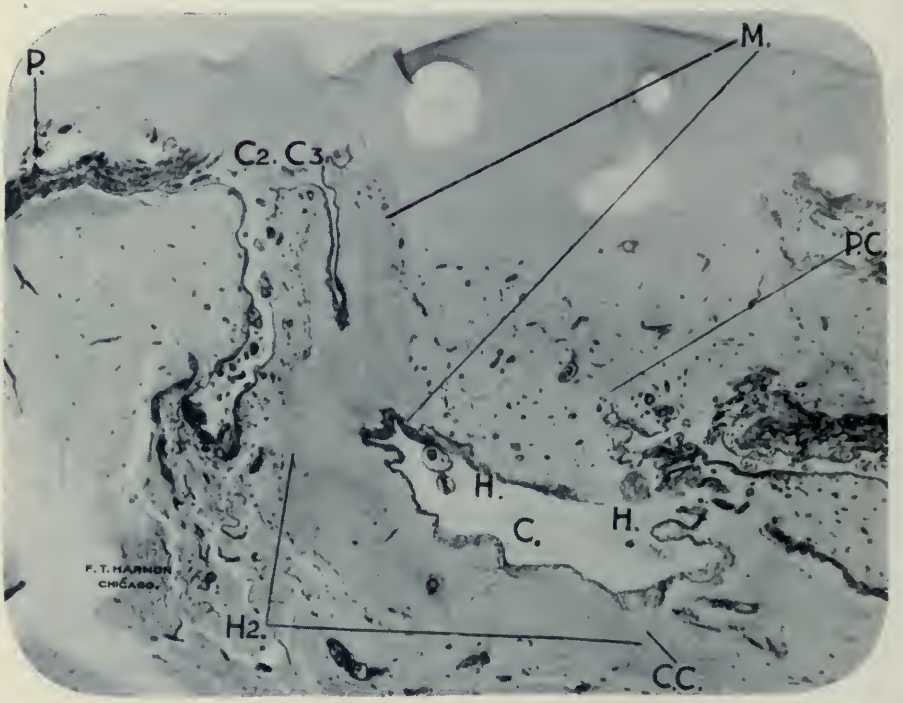

Fig. 1.—Syringomyelia cavities in the cervical cord enlargement. The main cavity (*C.*) contains patches of "homogenized" glia (*H.H.*) and two vascular islands. The accessory cavities (C_2C_3) distinctly show their membranes originating from the pia. H_2 is the zone of "hemogenization;" *P.c.*, posterior columns; *M.*, new forming membrane. Alzheimer-Mann stain; celloidin section × 20.

a granular mass in the center, part of which dropped out, leaving irregularly shaped cavities. The lower lumbar region and the entire sacral region presented slightly disfigured posterior columns, being otherwise well preserved.

The medulla, midbrain and the hemispheres revealed nothing pathologic.

Microscopic Examination.—The changes found in the spinal cord in general may be classified as (1) cavities, (2) thickened or hyperplastic pia, (3) a superabundance of vessels and capillaries enclosed within islands, and (4) so-called zones of homogenization.

The cavities showed well only in the cervical region, where six of them could be easily made out. Figure 1 shows three of the cavities; the largest, oblong and very irregular in shape, occupying the middle of the section behind the central canal and lined by a thick connective tissue membrane. The accessory cavities, to the left of the larger one, communicate with each other and reach the periphery of the spinal cord, where detachments of the pia can be seen running along their edges and forming a lining. The other three cavities,

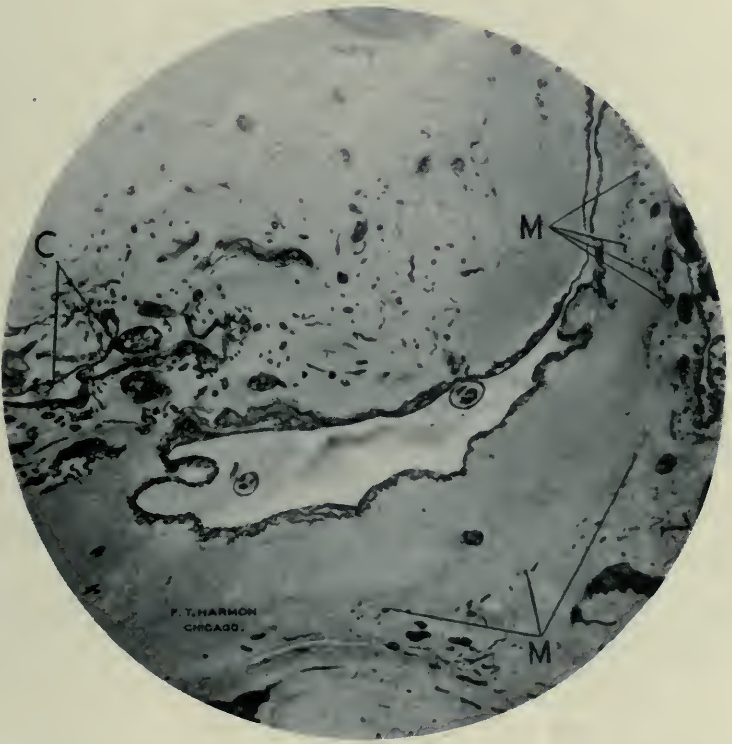

Fig. 2.—Part of the cervical enlargement of the spinal cord. The center is occupied by a large cavity lined by a collagenous connective tissue membrane. The larger portion of the cavity is filled with pale mass remnants of the homogeneous area surrounding the cavity and separated from the preserved parenchyma of the spinal cord by a thin irregular membrane, *M*. The upper left quadrant is covered with numerous vessels, vascular islands and remnants of a cavity, *C.*, lined by a connective tissue membrane. Alzheimer-Mann stain; celloidin section, × 23.

not shown in the picture, occupy the right portion of the posterior columns and the right posterior horn. Each cavity contained a powerful connective tissue membrane which did not show any epithelial lining, but was studded in some places with a great amount of vessels (Fig. 1, C_2). The cavities were separated from each other by a homogeneous substance, a pathologic glia, which was also present within the cavities themselves (Figs. 1 and 2). All the cavities ended blindly in the spinal cord and were in no way connected with the central canal, but communicated with the inner surface of the pia. Those portions of the posterior columns which were not taken up by the cavities were converted into a homogeneous mass (Figs. 1 and 2). The anterior horns were cut off by the latter, leaving intact the anterior portion, which was

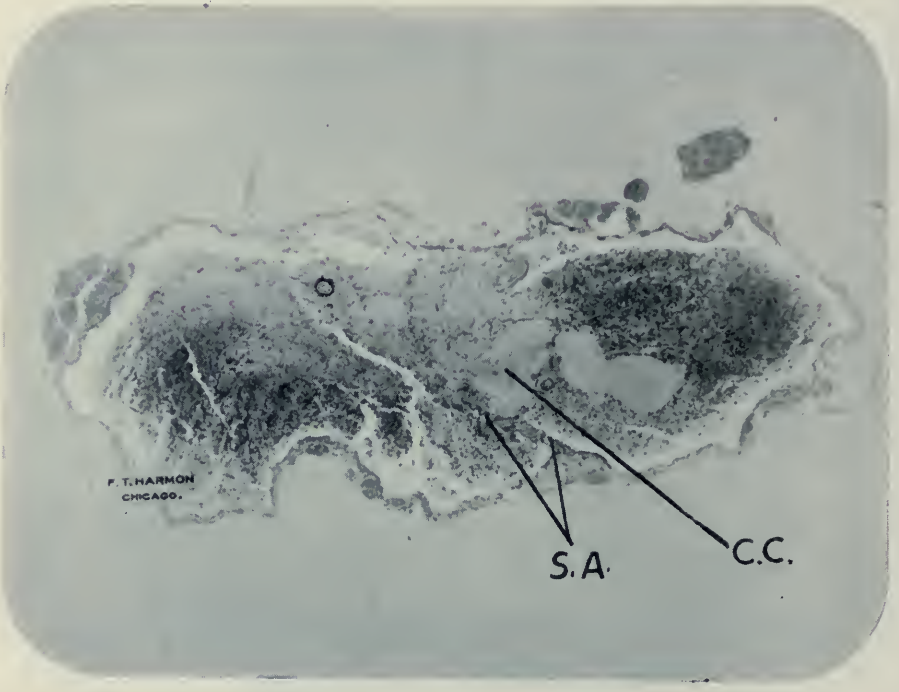

Fig. 3.—Dorsal segment in a case of syringomyelia. The thickened pia is torn off, the parenchyma is collapsed and disfigured. *S.A.*, anterior fissure filled with a thickened pia; *C.C.*, central canal. The lateral columns are well preserved; the broken down posterior half of the spinal cord contains pale islands of pathologic glia. Combined Weigert Pal-Van Gieson stain, × 11.

exceedingly vascular. The lateral and anterior, as well as the undamaged portions of the posterior columns, were more or less well preserved; but many nerve fibers in them were replaced by glia tissue which was much more abundant than in a normal cord. In the dorsal, lumbar and sacral portions there were no real cavities, the empty spaces to be encountered there being due to the falling out of the friable mass. The photomicrograph (Fig. 3) gives some idea of the changes in the dorsal region which was disfigured and collapsed. The lateral and anterior columns were somewhat preserved, while in the lumbar region (Fig. 4) the posterior columns showed an abundance of connective tissue strands, the subpial marginal glia zone being totally obliterated and the edge of the white matter depressed. Other sections of the dorsal region, partially reproduced in Figure 5, appeared uniform, were granular, and were occupied in some places by connective tissue fibers forming so-called islands. The tissue was very unstable, was friable, and was very losely held together, as if being prevented from falling apart by the surrounding powerful pia. Some portions of the spinal cord substance were missing, and an empty space was plainly in evidence.

In contrast to marked degeneration of the cord substance, the pia was remarkably developed. All along the spinal cord, especially in the cervicodorsal region, this membrane was greatly thickened. It gave a lining to the cavities (Fig. 2), even obliterating some of them and appearing distinctly fibrillar. In thin sections the pia fibers showed no nuclei, but a thicker section showed that the latter could be detected in the form of large spindle-shaped bodies with rounded edges, filled with chromatin granules. Young connective tissue cells, fibroblasts, perivascular infiltrations, plasma cells, lymphocytes, pigment deposits or hemorrhages were totally absent in the pia. The pia entirely enveloped the spinal cord, carrying a great number of hyperemic veins and capillaries. It invaded the spinal cord substance in the form of bands, and

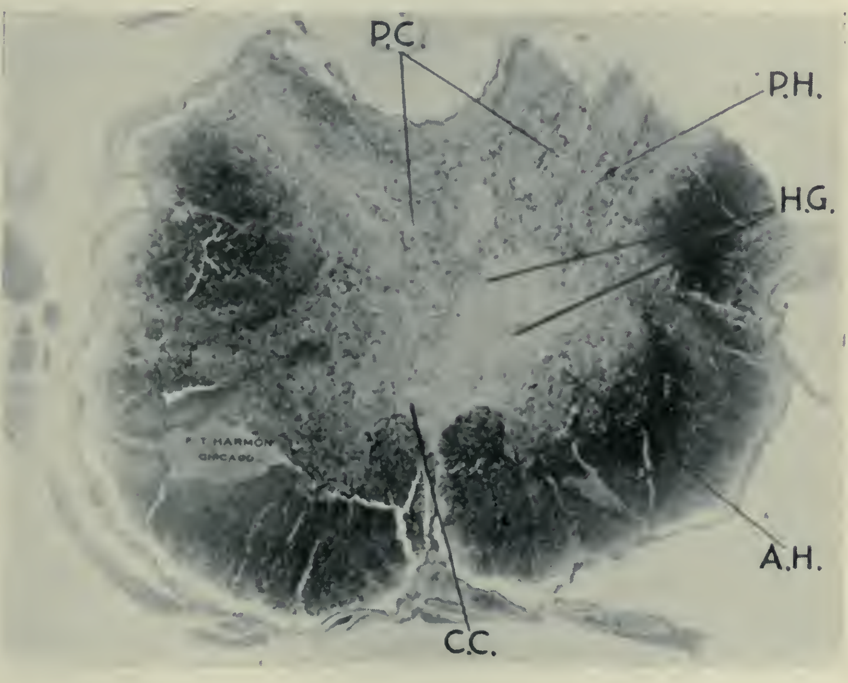

Fig. 4.—Lumbosacral region; the thickened pia forms in the posterior columns a marked concavity; the anterior and lateral columns are well preserved. *C.C.*, central canal; *A.H.*, anterior horn cut off from the posterior horn, *P.H.*, by a mass of homogeneous glia, *H.g.* Weigert-Pal stain counterstained with Van Gieson, × 11.

was closely adherent to the subpial glial tissue, but did not press on the posterior or anterior roots. We have here a hyperplastic pia, which, like a fibrous ring, surrounds the spinal cord, especially in places of its greatest destruction (cervicodorsal portions). The outlined features can very well be followed up with Van Gieson, Mallory-Jakob, Alzheimer-Mann, or light-green-fuchsin stains, but especially well with the combined Bielschowsky-Alzheimer method which will be described in detail at the end of this paper.

The vessels were exceedingly numerous in both gray and white substances, in the normal as well as in the pathologic areas. The homogeneous zone surrounding the cavity was rather poor in vessels, but the membrane lining the cavity was as a rule enormously vascularized (Figs. 1 and 6). Yet, even with Weigert's elastic stain, signs of vascular proliferation could not be found. Nor could any signs of inflammation be found, like perivascular infiltrations, distention of Robin-Virchow's lymph spaces in the adventitia, or any intima changes. The lumen was almost always open, containing some red cells, and was narrowed because of the greatly proliferated adventitia. This uniformly showed several layers of connective tissue fibers with few or no nuclei, staining very readily with Van Gieson, Alzheimer-Mann, Jakob-Mallory, and still better with a combined Bielschowsky-Alzheimer-Mann stain. With all these stains it was possible to demonstrate the hyperplastic state of the adventitia in every vessel and capillary to be found in the pathologic area. In the preserved portions of the anterior horns, and lateral and anterior columns, the vessels were hyperemic and in some places were surrounded by a thickened prolongation of the pia, but did not show hyperplastic adventitia. In some regions of the spinal cord, especially the gray matter and around the membranes of the cavities, the vessels, however small, were surrounded by connective tissue rings forming round or oval islands of various sizes. These are the so-called "encapsulations" of Petren[4] (Figs. 5 and 6), already pictured by Lockhart Clarke.[5] These islands or encapsulations sometimes contain several small vessels or capillaries with thickened adventitial walls, consisting, like the other vessels, of hyperplastic connective tissue. The islands were areolar in structure (Fig. 5), and consisted of meshes of glia fibers which were either empty or contained remnants of broken up glia tissue (nuclei, cell bodies and fragments of fibers). In some places the islands showed several concentric rings, or an island came into view that contained several separate rings, these being smaller vessels enveloped by a thick hyperplastic adventitia (Fig. 6). Such sclerosed vessels surrounded by one common capsule were described by Thomas-Hauser[2] as "formations conjonctivo-vasculaires," and by Cerletti[6] "vascular aggregations." Petren calls them vascular islands which he asserts originate from the encapsulations. The capsule comes, he thinks, from the bands of connective tissue invading the posterior horns. They grow so large that for lack of space they have to fold, enveloping portions of gray matter and forming rings enclosing gray matter.

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[4] Petren, Carl: Beiträge zur pathologische Anatomie und zur Pathogenese der Syringomyelie und der Syringobulbie, Virchows Arch. f. path. anat. **196:** 377, 1909.

[5] Clarke, J. Lockhart: On the Pathology of Tetanus, Med. Chir. Trans. **48**:255, 1865; Pathologic Investigations in a Case of Paraplegia, Brit. and Foreign Med. Chir. Rev. **33**:485, 1864.

[6] Cerletti, Ugo: Die Gefässvermehrung im Zentralnervensystem; Nissl-Alzheimer's Arb. **3**:1, 1911.

When the latter is absorbed, a vascular island remains. Instead of nerve tissue, we have, then, connective bands and sclerosed vessels—a scar as it were—representing the ultimate healing process of the diseased spinal cord tissue. On the other hand, this connective tissue proliferation accompanied by vascular sclerosis may appear as a forerunner of a progressively developing cavity, acting as a "destructive" agent. The destructive stage is followed by a "constructive," progressing, healing process which takes place after the parenchyma has been destroyed. The connective tissue proliferation is, according to Petren, a chronic inflammation which results in the formation of numerous sclerosed vessels. Such cases Petren calls sclerotic syringomyelia in distinction from the glial type (syringomyelia gliosa) which is due to hyperplasia of the glia. The sclerotic type of syringomyelia runs a shorter clinical course; the latter, a more protracted one. Such are some of the conclusions Petren arrived at from the study of connective tissue bands, connective tissue islands and encapsulations.

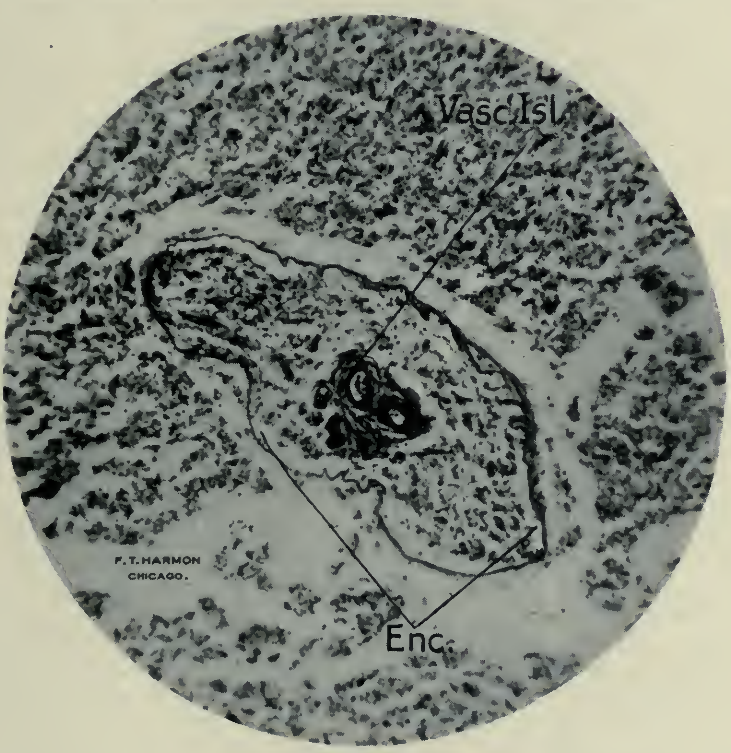

Fig. 5.—Lower dorsal region; the upper portion is granular; the lower, greatly rarefied and in some places devoid of substance. The black dots are cross sections of axons; the paler subtsance is glia tissue. The middle of the illustration is occupied by an "encapsulation" (*Enc.*), the membrane of which is separated from the vascular island (*Vasc. Isl.*) by decomposed glia tissue and remnants of nerve fibers. Bielschowsky stain; paraffin section, × 100.

As these three types were represented in my case, Petren's interpretations are not in complete accord with my findings. The photomicrographs show the connective tissue formations are present only in those places where there are to be found remnants of a substance described under various names (couches scleroses by Hallopeau,[7] colloid substance by Roth,[8] "Gewebsmasse" by Chiari,[9] homogeneous substance by Schwarz[10] "homogenization" zone by Schlesinger,[1] primitive gliosis by Phillippe and Oberthür[3]). This substance is especially marked in Photomicrograph 2 in the shape of a broad mass surrounding the membrane. This mass can be found within the cavity itself as a patch sticking to the wall, or between two adjacent membranes (Fig. 1) or within the meshes of the latter, but is never found beyond the outer wall of the membrane. The latter thus cuts the glia or the parenchyma into two parts, the inner part, pathologic; the outer, normal.

The microscopic examination of the homogeneous masses showed a network of delicate glia fibers, with a number of small glia nuclei more or less well preserved, though some stained rather badly. Quite often there was but a remnant or a fragment of a glia reticulum with a great mass of uniformly stained, usually pale, round bodies of various sizes. Some of them gave a perfect reaction for amyloid, and there can be no question that many of these round or oval formations were amyloid bodies. Mixed with the latter, there were other spheroid bodies of almost similar size, equally stainable with various anilin dyes, but containing a very small, shrunken nucleus, apparently collapsed, always located at the very periphery of the cell body, as if ready to leave it (Fig. 7). In some such cells half of the nucleus could be seen as if sticking out of the cytoplasm, and some cells were encountered without any nucleus whatever. Such cells greatly resemble amyloid bodies, from which they differ by failing to show the iodin reaction. It is probable that some such cells ultimately become actual amyloid bodies, in accordance with the views of Fromman, Obersteiner, Redlich[11] and others who consider them as the products or modifications of glia. In addition, there is in the homogeneous substance a third variety of spherical globules, very numerous and much smaller and paler, appearing as fine, minute droplets. They are especially numerous within the cavities, in the walled-off, or as Thomas and Hauser[12] express it, in the areas "parceled out" by connective tissue bands and vessels. They are also to be found in great masses within the connective tissue spaces, in the adventitial walls, in the encapsulations or islands, in the pia meshes, in short, wherever the glia is destroyed or damaged. One gains the impression that the proliferating connective tissue in spreading to the periphery envelops them as a useless substance, before they finally perish and become absorbed. The remarkable feature is that even in these greatly changed patches of glia tissue, there may be found well preserved nerve fibers, or,

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7. Hallopeau, H.: Contributions à l'étude de la sclérose diffuse péri-ependymaire, Soc. de Biologie, Ser. 5, **21**:169, 1869.

8. Roth, W.: Gliome diffuse de la moelle, syringomyélie, atrophie musculaire, Arch. de physiol. normale et pathol. **10**:612, 1878.

9. Chiari.: Ueber die Pathogenese der sogenanten Syringomyelie, Ztschr. f. Heilk. **9**:307, 1888.

10. Schwarz: Ein Fall von Meningo-myelitis Syphilitica mit Höhlenbildung im Rückenmark, Ztschr. f. klin. Med. **34**:467, 1898.

11. Redlich, E.: Die Amyloidkörperchen des Nervensystems, Jahrb. f. Psychiat. u. Neurol. **10**:1, 1891.

12. Thomas, A., and Hauser, G.: Histologie pathologique et pathogénie de la syringomyélie, Nouv. iconogr. de la Salpêtrière **17**:376, 1904.

what is more frequent, fragments of well-stained myelin and axons. These changes in the "homogenized" area can be much better followed up in longitudinal section (Fig. 7). The photomicrograph distinctly shows the nerve fibers, enlarged or ampule-like, in some instances thinned, of distinct fibrillary structure and staining well. In some fibers the axon is plainly visible; in others, it is masked by a powerful myelin sheath forming basket-like enlargements connected with each other by thin strands of fibrils. The spaces between adjacent fibers is occupied by the round bodies and spherules described above, the entire mass resembling Alzheimer's "Füllkörperchen" and his ameboid glia. Places less damaged or not much disintegrated show a few poorly stained gitter-cells, large protoplasm-rich glia cells uniformly stained, with an eccentric nucleus, and occasionally even so-called myelophages. Herxheimer stain, which was hard to carry out on account of the difficulty of obtaining proper frozen sections of a friable granular mass, showed the presence of fat globules.

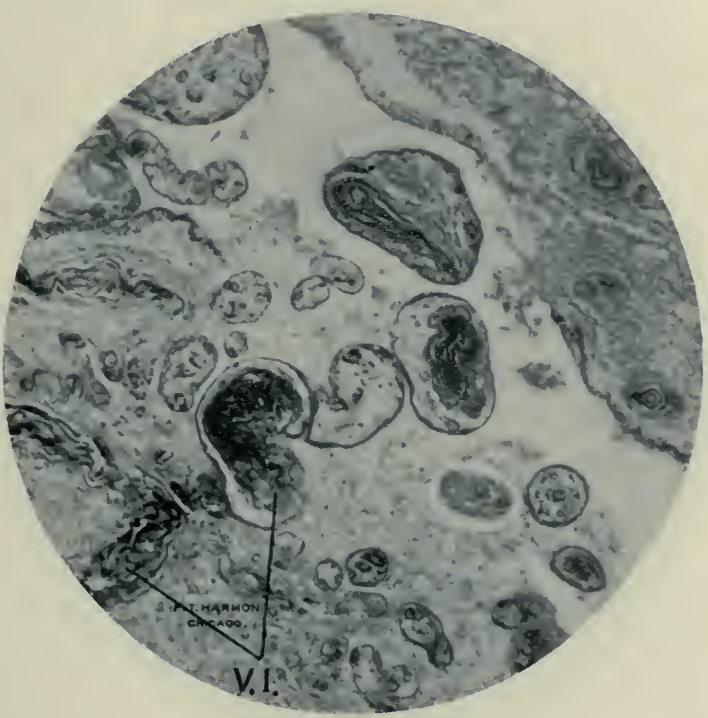

Fig. 6.—Vascular islands, *V.I.;* the rest of the round formations are encapsulations, some inclosing longitudinally cut vessels; from an area near the cavity. Combined Bielschowsky-Alzheimer-Mann stain; paraffin section, × 85.

The central canal was found occluded by a mass of proliferated cells, nowhere connected with the cavities, and surrounded by a homogeneous mass which microscopically (Fig. 8) appeared to consist of a delicate glia reticulum containing a number of glia nuclei. It somewhat resembled the structure of the "homogenization" zone, but the process evidently had not progressed very far. The lateral and anterior columns and the preserved gray matter of the anterior horns showed no gross abnormalities, like ganglion cell changes, secondary degenerations, etc. The medulla and cortex exhibited vascular changes only. In the former, some vessels were slightly infiltrated with lymphocytes. In the cortex there was marked proliferation of capillaries, without any other changes, like infiltrations, ganglion, glia or meningeal changes. Pathologic changes were exclusively confined to the spinal cord, being at some levels more pronounced, at others less, confined principally to the glia, the vessels and pia, with or without cavity formation. It is obvious that the latter is not an essential factor in syringomyelia, as many levels in this case did not show cavities at all. Probably they would have formed had the patient lived longer; that is, the changed areas which appeared as homogeneous zones would ultimately have become transformed into cavities.

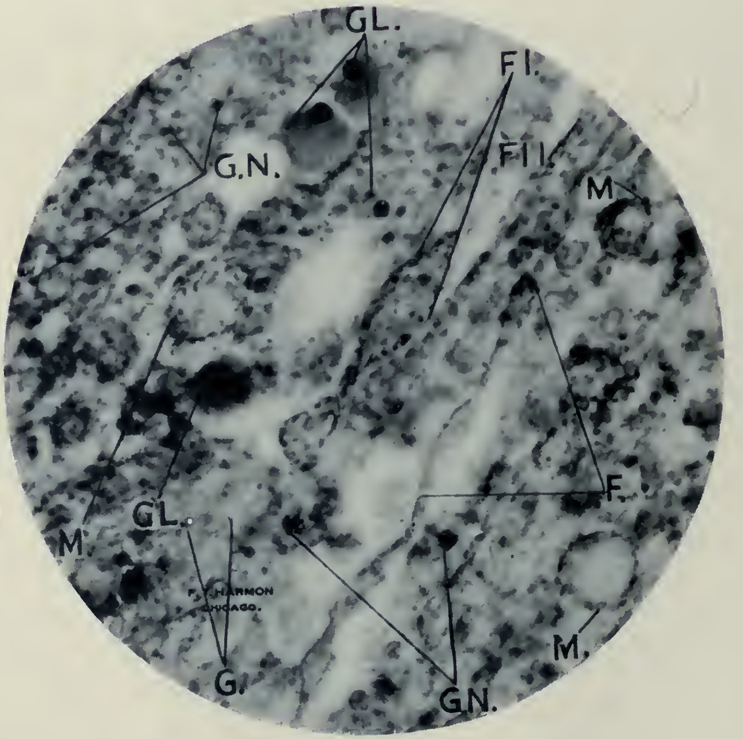

Fig. 7.—Longitudinal section of the "zone of homogenization." *G.n.,* glia nuclei; *Gl,* glia cells with picnotic nuclei about to desert the homogeneous cell bodies; *M,* myelin; *Fi,* nerve fiber with granular ampular enlargements of the myelin sheath; *F* and *Fii* comparatively less changed nerve fibers; *G,* sale glia cells resembling amyloid bodies. Combined Bielschowsky-Alzheimer-Mann stain, × 700.

Of the five factors (changes in the pia, the vessels, areas of homogenization, island and membrane formation (Petren's encapsulations), the most important, the primary factor, is change in the glia. But the character and nature of these glia changes are quite different from those to be found in various other spinal cord lesions, like multiple sclerosis, secondary degenerations, myelitis, amyotrophic lateral sclerosis and subacute combined cord degeneration, in which glia changes are also the dominant feature. For in syringomyelia the glia does not show the exquisite progressive or proliferative properties so pronounced in the former diseases, nor the equally exquisite regressive character seen in myelitis. It is true that in syringomyelia there may be encountered some signs of a proliferative nature, represented by a few gitter-cells, fat globules, few myelophages, and large protoplasmic glia cells; but these signs are so insignificant, so scarce, and so overshadowed by the opposite phenomena of retrogressive character, outlined above, that at least in this case we cannot speak of a proliferation of glia.

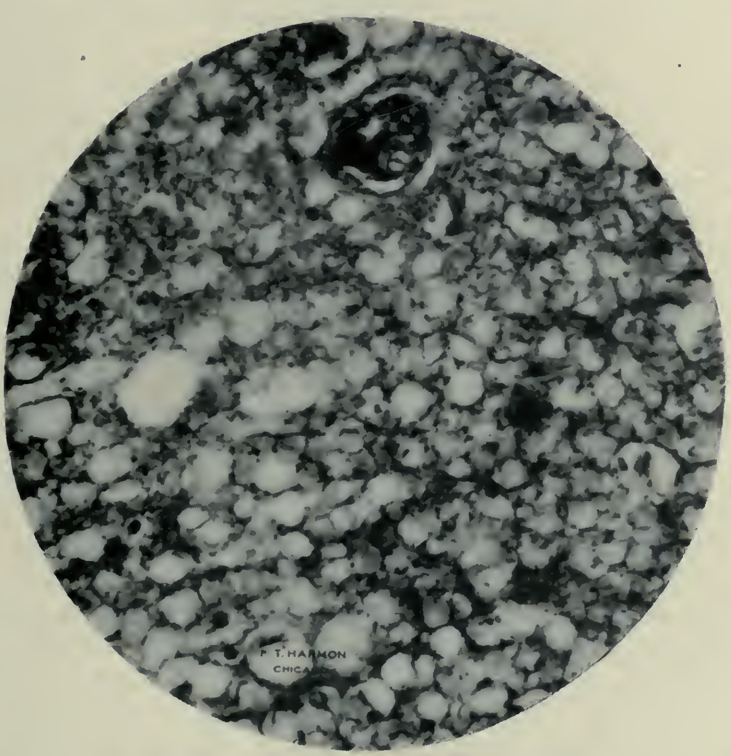

Fig. 8.—An area from the posterior commissure consisting of a glia reticulum, glia nuclei (black dots) and an aggregation of vessels (in the upper portion of the illustration). Combined Bielschowsky-Alzheimer-Mann stain; paraffin section, × 450.

On the contrary, the glia is rather regressive in character, especially near the cavities where the process must be assumed to be greatly advanced, while in the lower dorsal regions the morbid process is evidently of more recent date, no cavity formation being present. Furthermore, if glia tissue does proliferate, as in multiple sclerosis, subacute combined degeneration or secondary degenerations caused by cerebral lesions (capsular or encephalitic lesions), the proliferation obtains at the expense of destroyed nerve tissue. The latter, however, is in syringomyelia, as shown by my case, not degenerated. Even in the most damaged areas, like the interior of the cavities themselves, nerve fiber fragments with their myelin and axon can be seen comparatively undamaged; that is, not enveloped by the glia cells, nor converted into lipoid substances, harbored by numerous and various gitter-cells, as is the case when the glia undergoes real proliferation. Nor are the glia changes in syringomyelia of the type seen in myelitis, for instance, where it assumes the ameboid features.

COMMENT

Evidently the syringomyelic changes of glia are altogether different from those to be found in progressive or regressive states, a fact already noted by Schultze[13] in 1882, when he coined for these glia changes in syringomyelia a new name, that of gliosis. Ten years later, Hoffman[14] called it primary, central, gliosis. To make clear the meaning of gliosis, Schultze defined it as the mildest or milder stage of glioma formation, as the forerunner of gliosis. In a later publication[15] he compared its relationship to glioma as that of "a hill to a mountain." Bernstein and Horwith[16] express themselves still more indefinitely. "There are seen," they say, "areas of gliosis which perhaps may bear toward the tissue we call glioma a relation similar to that which a fibroma bears to sarcoma (leaving out of consideration the question of malignancy)." Thus, according to Schultze and others, the proliferated glia in syringomyelia is different from a proliferated glia in other spinal cord diseases. If glia proliferates and breaks down it is called gliosis; if it proliferates and does not break down (multiple sclerosis, for instance) then it is not a gliosis. According to modern neuropathologic research, such a distinction between the two types of a proliferative glia is wholly untenable; in fact, it is incomprehensible. From Schultze's publications and his numerous followers (Miura,[17] for instance) the sole outstanding, undoubted fact remains that in syringomyelia we have to deal with a primary lesion of glia, either around the central canal, or in Rolando's substance, or in some other part of the spinal cord. This fact is absolutely established, and we therefore must come to the conclusion that syringomyelia is essentially a disease of the glia and not of the nerve tissue, like multiple sclerosis, amyotrophic lateral sclerosis, subacute combined degeneration, etc., which are essentially parenchymatous nerve lesions, in which the glia is secondarily involved.

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[13] Schultze, L.: Beiträge zur Pathologie und pathologische Anatomie des centralen Nervensystems, Virchows Arch. f. path. Anat. **87**:510, 1882.

[14] Hoffman, J.: Zur Lehre von der Lehre von der Syringomyelie, Deutsch. Ztschr. f. Nervenh. **3**:1, 1892.

[15] Schultze, F.: Die Pathogenese der Syringomyelie, Compt. rend. du 12me Congrés Int. de Med. à Moscou **4**:265, 1897.

[16] Bernstein, E. P., and Horwith, S.: Syringomyelia with Pathological Findings, Med. Rec. **84**:698, 1913.

[17] Miura, K.: Ueber Gliom des Rückenmarks und Syringomyelie, Beit. z. path. Anat. u. z. allg. Path. (Ziegler's Beitr.) **11**:91, 1892.

The glia changes must necessarily be exceedingly chronic in course, as many cases last from forty to fifty years. In fact, the beginning of the trouble can never be definitely asserted, as the patient seeks advice only when he is much discomforted by some motor, trophic or other disturbance, and may go around for years without complaining, yet harboring the disease. This was probably the case with my patient, who gave a history of but three weeks' duration, and for some symptoms, two days. The deepest and most advanced destruction was in the dorsal spinal cord which suggests the possibility of his having some objective sensory disturbances on the trunk, chest, lumbar region, etc., which did not trouble him in any way until they became complicated by trophic lesions and forced him to go to the hospital. The process in every case must be much older than the date of the patient's first complaints, probably extending, as Saxer[18] justly remarks, to the embryonal state, and probably being inborn. In other words, a person suffering from syringomyelia is probably born with a deficient glia, just as a person with progressive muscular atrophy or dystrophy is born with deficient ganglion cells or deficient muscle fibers; in both we have to deal with a developmental defect, an abiotrophy. The abiotrophy, in the former, shows itself in glia cells; in the latter, in the nerve or muscle cells.

As in the latter a great many ganglion cells are usually undamaged, appearing healthy, so in the former (syringomyelia) a great many glia cells may escape destruction and may even exhibit signs of increased activity, in the form of a rich cytoplasm, formation of myelophages, gitter-cells, etc. As in progressive muscular atrophy, in which the deficient ganglion cells may be confined to certain levels or segments of the spinal cord, the cervical region, for instance, in syringomyelia, the deficient glia also may be confined to a certain level, sparing the rest. When the deficient glia finally breaks down, disintegrates, it becomes a useless foreign body, which like any other foreign body becomes enveloped or walled off by connective tissue. The connective tissue proliferation is thus, contrary to the assertions of Hauser, Sterling[19] and others, a secondary process, obtaining only when glia destruction is at its maximum, originating, as my case shows, in the pia and partly from the vessels. It is of hyperplastic, proliferative, not inflammatory character, and may be looked on as a natural reaction to the pathologic process displayed in the cavity. The latter is filled with fluid, and is usually greatly distended, the distention of course affecting the pia greatly.

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[18] Saxer, F.: Beit. z. path. Anat. u. z. allg. Path. (Ziegler's Beitr.) **32:** 276, 1902.

[19] Sterling, W.: Beiträge zur Lehre von der Morvanschen Krankheit, Ztschr. f. klin. Med. **56:**474, 1905.

I had an opportunity to witness such a distention during an operation (by Dr. Karl Meyer) on a patient with syringomyelia symptoms, which I had reason to believe were probably due to an intraspinal tumor. On opening the dura in the lower cervical region, the cord appeared distended, sausage-like, very pale, and without any signs of pulsation. When the posterior columns were incised, fluid came out with great force; the spinal cord collapsed at once, became hyperemic, and immediately showed active pulsations. The enormous pressure sustained by the cord in syringomyelia must be counterbalanced by the surrounding meninges, especially the pia, which is called on to hold the fluid and the remnants of the broken up cord firmly together, the hyperplasia being the ultimate result of this increased function. The same is true of the blood vessels, the adventitial walls of which are always greatly thickened, hyperplastic, thus protecting them from a too increased pressure and a probable total occlusion. This vascular state is absolutely pathognomonic of syringomyelia, as in no other pathologic state could I see anything like it, and it is also pointed out without exception in every description of syringomyelia. Very often the sclerosed vessel is the center of various islands, encapsulations, which, as I have already stated, resemble in their structure, mode of growth, etc., the "homogenization" zone around the cavity.

If the glia is deficient from earliest life, then apparently normal spinal cords must be met which would show an abnormal glia state in the vicinity of the central canal or in the posterior horns, the usual seats of syringomyelia. This is actually the fact. Thus Schlesinger and Obersteiner[20] saw many spinal cords, otherwise normal, showing a wider and larger posterior commissure, protruding into the posterior columns in the form of bands or massive processes and consisting of glia meshes, vessels and even occasional areas of homogenization or of fully developed gliosis. Zappert[21] found in a spinal cord of a 19-months-old child a dilated spinal canal, glia proliferation, in which a homogeneous zone surrounded the central canal. These facts demonstrate the correctness of the assumption that syringomyelia dates back to the earliest days of existence and may run through the entire life unobserved or undiscovered; in short, that syringomyelia is a developmental anomaly. But this developmental anomaly does not pertain to a patent central canal (Virchow,[22] Leyden[23]) or to a broken down tumor (Simon,[24] Westphal[25]) or to slumbering epithelial cells around a maloccluded central canal which suddenly wake up and proliferate (Hoffman[14]), or to a proliferation and subsequent breaking down of glia cells (Schultze[13, 15]), but pertains to the inborn deficiency of glia tissue as outlined above. Nor has syringomyelia anything to do with traumatic and nontraumatic hematomyelias (Minor,[26] Pitres and Sabrazes[27]), myelitis (Hallopeau,[7] Joffroy-Achard[28]), pachymeningitis or leptomeningitis (Rosenblath[29]), all these morbid conditions having been advocated as the possible causes of syringomyelia. They all may produce cavities in the spinal cord, but not syringomyelia, which differs from the enumerated morbid states anatomically and histologically. Only those spinal cavities should be referred to as syringomyelia which exhibit glia or other changes as represented, in part, by the appended photomicrographs.

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[20] Schlesinger, H.: Die Syringomyelie, Ed. 2, 1902, p. 324.

[21] Zappert, J.: Kinderrückenmark und Syringomyelie, Wien. klin. Wchnschr. **14**:949, 1901.

STAINING METHODS

The staining methods must necessarily vary according to the variety of tissues encountered in this case; axons, myelin, glia tissue, gray matter, vessels and pia. Of the numerous methods used, the most reliable and satisfactory one proved to be that of Bielschowsky counterstained with Mann's mixture (methyl blue-eosin). I had used it very succesfully on frozen sections in my studies of subacute combined cord degenerations,[30] while in this case frozen sections could not be used very well on account of the great disintegration of the tissues. I was therefore compelled to use paraffin sections of tissues that were fixed in chrom-fluorine and potassium bichromate (Weigert's

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22. Virchow, R.: Die Betheiligung des Rückenmarks an der Spina Bifida und die Hydromyelie, Virchows Arch. f. path. Anat. **27**:575, 1863.

23. Leyden, E.: Klinik der Rückenmarkskrankheiten, **2**:447, 1875.

24. Simon, T.: Beiträge zur Pathologie und pathologische Anatomie des Centralnervensystems, Arch. f. Psychiat. **5**:108, 1875.

25. Westphal, C.: Ueber einen Fall von Höhlen-und-Geschwulstbildung im Rückenmark mit Erkrankung des verlängerten Markes, Arch. f. Psychiat. **5**: 90, 1875.

26. Minor, L.: Zur Lehre von der Syringomyelie, Ztschr. f. klin. Med. **34**: 373, 1898.

27. Pitres and Sabrazès: Contributions a l'étude clinique et anatomie pathologique de l'hematomyélie centrale, Compt. rend. du Congrés Int. à Moscou **1**:292, 1897.

28. Joffroy, A., and Achard: De la myélite cavitaire, Arch. de Physiol. normale et pathol. **10**:435, 1887.

29. Rosenblath, W.: Zur Casuistik der Syringomyelie und Pachymeningitis cervicalis hypertrophica, Deutsch. Arch. für klin. Med. **51**:210, 1893.

30. Hassin, G. B.: Histologic findings in two cases of subacute combined cord degeneration, Med. Rec. **91**:885, 1917.

mordant fluid). After removal of paraffin the sections are stained with Bielschowsky's method in the usual manner. Without being impregnated with gold they are counterstained with Alzheimer-Mann (1 per cent. phospho-molybdic acid) for one hour, followed, after the sections are twice washed in distilled water, by methyl-blue-eosin for one half hour or longer. The pictures are strikingly beautiful, the connective tissue, the glia, the axons, myelin, the gray matter, all staining in different colors as shown in the appended colored reproduction.

31 North State Street.

ANALYSES OF BLOOD OF INSANE PATIENTS

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WARREN, PA.

Ishida[1] reported a deficiency of chlorin in the blood of dementia praecox patients and suggested the use of sodium chlorid solution intravenously in the treatment of this condition. He estimated the chlorin values of the blood of five untreated cases of dementia praecox, and the amounts varied from 0.199 to 0.220 per cent. The analyses were made by Professor Horinchi by the Vollhard-Salkowski method. Guthrie[2] published a report of the results of the treatment of fifteen cases of dementia praecox based on chemical analyses of the blood and reported a deficiency of chlorin, but the quantities found and the methods of analysis are not mentioned. Rappleye[3] stated that in some cases of catatonia he found a deficiency of chlorin. Miller[4] quoted analyses made by Weston of the blood of ten cases of dementia praecox, the chlorin being normal in each case, and he stated that "there was no deficiency of chlorin in the blood . . . in any of fifteen other cases examined." Rappleye[3] studied the blood urea in catatonic patients at from three to seven day intervals before, during and after the catatonic state. He found that a marked drop, about 50 per cent., in the urea nitrogen occurred early and rapidly in catatonia.

The figures of Ishida, Rappleye and Weston do not agree. In Rappleye's cases the urea was determined when catatonia was present, as well as when it was not. Weston's figures are from one analysis made when the patient was not in the catatonic state.

Since there is some disagreement in the published estimates of chlorin and urea nitrogen, the accompanying tables may be of interest. They represent analyses of the blood of thirty insane patients, selected cases of dementia praecox, epilepsy and manic-depressive insanity. The analyses were made a little more than two years ago. All the patients were living under practically identical conditions and had substantially the same diet. As the blood was collected before breakfast, twelve hours after the preceding meal, the effect of diet may be disregarded. Care was taken to use clear cases, and in each case selected there was unanimity of opinion when the patient was pre-

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1. Ishida, N.: Am. J. Insan. **73**:541 (Jan.) 1917.
2. Guthrie: Dementia Praecox Studies **1**:61, 1918.
3. Rappleye, W. C.: J. Nerv. & Ment. Dis. **49**:130 (Feb.) 1919.
4. Miller, W. C.: Arch. Neurol. & Psychiat. **1**:313 (March) 1919.

sented at a staff meeting for diagnosis. The Wassermann reaction in all cases was negative (cholesterolized human heart antigen), and no abnormal substances were found in the urine of any patient.

TABLE 1.—Blood Analyses of Ten Cases of Manic Depressive Insanity

			Milligrams per Hundred C.c. of Blood								
Number	Age	Total Nitrogen, Percentage	Nonprotein Nitrogen	Uric Acid	Urea Nitrogen	Creatinin	Creatinin plus Creatin	Glucose	Chlorin as Sodium Chlorid	Calcium	Remarks
1	33	3.85	36	2.12	24	2.15	8.90	90	480	8.0	Hypomanic
2	40	3.45	33	2.60	16	1.90	7.60	90	490	7.8	Hypomanic
3	38	3.60	34	1.95	22	2.10	8.40	100	500	7.0	Hypomanic
4	29	3.55	33	2.20	20	1.85	7.20	110	510	9.1	Depressed
5	40	3.20	31	2.40	20	1.75	7.60	120	506	8.8	Depressed
6	36	3.45	33	1.80	24	1.60	7.40	90	490	7.7	Depressed
7	29	3.60	33	2.20	27	2.00	8.00	85	484	8.8	Depressed
8	37	3.70	35	2.10	24	2.10	7.90	90	480	8.4	Interval
9	40	3.60	34	2.30	22	1.90	8.00	90	490	8.0	Interval
10	44	3.75	32	2.75	20	1.80	7.00	110	510	7.8	Interval

TABLE 2.—Blood Analyses of Ten Epilepsy Cases

			Milligrams per Hundred C.c. of Blood								
Number	Age	Total Nitrogen, Percentage	Nonprotein Nitrogen	Uric Acid	Urea Nitrogen	Creatinin	Creatinin plus Creatin	Glucose	Chlorin as Sodium Chlorid	Calcium	Remarks
11	35	3.16	32	1.90	18	1.25	6.20	100	480	7.8	Grand mal
12	32	3.52	36	1.85	20	2.25	8.20	90	495	7.8	Grand mal
13	28	3.48	34	2.20	22	2.31	9.40	110	506	8.8	Grand mal
14	31	3.21	37	2.16	20	1.95	7.60	105	512	8.0	Grand mal
15	31	3.75	36	2.30	21	1.70	8.40	121	500	7.7	Grand mal
16	35	3.64	32	2.00	22	1.67	7.80	95	484	7.0	Grand mal
17	40	3.80	33	1.85	24	2.00	8.10	90	510	7.5	Grand mal
18	37	3.72	34	1.90	20	2.20	8.20	110	490	6.9	Grand mal
19	22	3.69	32	1.76	21	2.12	8.00	120	486	7.6	Grand mal
20	28	3.71	38	2.00	24	2.10	7.95	100	495	7.9	Grand mal

No seizures were recorded for forty-eight hours preceding the analyses.

TECHNIC

Blood was first drawn for the usual routine Wassermann reaction, and then at least 30 c.c. were collected in a tube containing 0.45 gm. of powdered, calcium-free sodium citrate. Oxalate was not used because calcium determinations were to be made with the blood. Analysis was begun at once, and four specimens were examined at the same time, this being as large a number as could be handled without an assistant.

The blood was divided as follows:

Ten c.c. of blood were added to 30 c.c. of 6.25 per cent. trichloracetic acid solution (freshly prepared) in a 50 c.c. volumetric flask.

The mixture was well shaken, distilled water was added until the solution reached the mark, and the flask was set aside for one hour. At the end of this time, the contents were poured into a tube and centrifugalized. The supernatant fluid was pipetted off or filtered through a double-washed filter paper and used for the determination of chorids by titration with potassium thiocyanate, nonprotein nitrogen by the method of Folin and Denis,[5] and calcium by Lyman's method.[6] The use of trichloracetic acid did not affect the chlorin values. Blanks were run each day for calcium and chlorin.

TABLE 3.—Blood Analyses of Ten Dementia Praecox Cases

			Milligrams per Hundred C.c. of Blood								
Number	Age	Total Nitrogen, Percentage	Nonprotein Nitrogen	Uric Acid	Urea Nitrogen	Creatinin	Creatinin plus Creatin	Glucose	Chlorin as Sodium Chlorid	Calcium	Remarks
21	21	3.52	31	2.40	16	2.00	8.10	105	515	5.7	Hebephrenic
22	18	3.75	33	2.60	17	1.82	7.90	101	510	5.5	Hebephrenic
23	28	3.44	34	1.50	22	1.90	8.00	106	510	6.2	Hebephrenic
24	21	3.26	33	1.90	19	2.16	7.20	111	494	8.0	Catatonic
25	36	3.64	31	2.00	16	2.20	10.00	120	491	8.0	Catatonic
26	27	4.00	39	2.60	22	1.85	9.40	112	506	7.8	Catatonic
27	23	4.00	40	2.70	18	2.10	8.10	105	512	7.8	Catatonic
28	26	3.80	33	1.90	16	1.90	8.66	124	500	6.0	Paranoid
29	29	3.25	33	2.00	16	1.70	9.00	117	500	8.0	Paranoid
30	32	3.19	33	1.80	18	2.15	9.21	109	515	6.5	Paranoid

Blood was not drawn during catatonia.

TABLE 4.—Normal Values *

	Author		
	Gettler and Baker[8]	Folin and Denis[19]	Myers and Fine[20]
Total nitrogen	3.7	....	3.0
Nonprotein nitrogen	30 - 45	22 - 37	25 - 30
Uric acid	1 - 3.5	0.7 - 3.7	1 - 2
Urea nitrogen	15 - 25	12 - 27	12 - 15
Creatinin	0.1 - 0.5	1.1 - 1.4	1 - 2
Creatin	3 - 6.5	5.2 - 8.1	5 - 10
Glucose	50 - 120	90 - 100[14]	90 - 130[15]
Chlorin as sodium chlorid	450 - 500	490[16]	457[17]
Calcium as Ca	5.2 - 9.6[18]		

* Data for this table were taken from the paper of Gettler and Baker. Total nitrogen is expressed in per cent. All other values are in terms of milligrams per hundred c.c. of blood. The figures for calcium are from Lyman's paper.

Five c.c. of blood were used for the determination of creatin, creatinin and glucose by the methods of Myers and Fine.[7]

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5. Folin, O., and Denis, W.: J. Biol. Chem. **26**:473 (Sept.) 1916.
6. Lyman, H.: J. Biol. Chem. **29**:169 (March) 1917.
7. Myers, V. C., and Fine, M. S.: Chemical Composition of the Blood in Health and Disease, Post-Graduate **30**:39, 1915.

One c.c. of blood was used for the urea determination, and one c.c. for control. Dunning urease tablets were used for fermentation, and the ammonia was determined by Nesslerization after aeration.

One c.c. of blood was added to 99 c.c. of ammonia-free distilled water and 1 c.c. of the dilution used for the determination of total nitrogen by the method of Folin and Denis.[5]

The remaining 10 or more cubic centimeters were used for the determination of uric acid by the method of Myers and Fine.[7]

The Kober colorimeter and nephelometer were used throughout and all glassware was certified.

The creatinin values recorded in the tables are far above the normals given by Gettler and Baker,[8] and Gettler.[9] For the method employed, however, the values are not above normal. The picric acid used answered the requirements laid down by Folin and Doisy,[10] and the sodium citrate, in the quantities used, had no bleaching effect.

Cholesterol is not included in the tables. Several hundred estimations have been made, but the results did not vary from the normal. Some of the results have already been published.[11]

The relation of the sugar content of the blood and spinal fluid was reported some years ago.[12] In sixty-five cases, the spinal fluid sugar averaged 60 per cent. of the blood sugar. Recently, Myers and Fine[13] found in fifteen cases that the spinal fluid sugar averaged 57 per cent. of that in the blood.

SUMMARY

The blood of epileptic, dementia praecox and manic-depressive patients shows no deviation from the normal content of total nitrogen, nonprotein nitrogen, uric acid, urea, creatinin, creatin, glucose, chlorin or calcium.[14]

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[8] Gettler, A. O., and Baker, W.: J. Biol. Chem. **25**:211 (June) 1916.

[9] Gettler, A. O.: J. Biol. Chem. **29**:47 (Feb.) 1917.

[10] Folin, O., and Doisy, E. A.: J. Biol. Chem. **28**:349 (Jan.) 1917.

[11] Weston and Kent: J. M. Research **26**:531, 1912.

[12] Weston, P. G.: J. M. Research **35**:199 (Nov.) 1916.

[13] Myers, V. C., and Fine, M. S.: J. Biol. Chem. **37**:239 (Feb.) 1919.

[14] In addition to the references given, the following papers may be of interest: Lewis and Benedict: J. Biol. Chem. **20**:61, 1915; Michaelis: Biochem. Ztschr. **59**:211, 1916; McLean and Vanslyke: J. Biol. Chem. **21**:361, 1915; Rogee and Fritsch: Biochem. Ztschr. **54**:53, 1913; Lyman, H.: J. Biol. Chem. **30**:1 (May) 1917; Folin and Denis: J. Biol. Chem. **14**:29, 1913; Myers, V. C., and Fine, M. S.: Post-Graduate, New York **29**:473, 1914.

SPLINTS USED FOR PERIPHERAL NERVE CASES AT THE U. S. ARMY GENERAL HOSPITAL NO. 11

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BOISE, IDAHO

Shortly after patients from overseas began to arrive at the U. S. Army General Hospital No. 11, it was decided that the splints which had been applied to the nerve cases had numerous disadvantages. They were heavy and cumbersome, and in a large number of cases were retarding rather than aiding recovery. With these faults in mind, each lesion was studied from the standpoint of splints, and a special group of splints was designed for each type of case. These were then tried out and the one found, by actual practice, to be the most satisfactory was adopted as a standard splint for a given lesion. The following descriptions, pictures and diagram cover the splints now in use.

SPLINT FCR FOOT DROP

This splint has been adopted as a standard splint for patients who are able to wear shoes, for it is sightly, simple and servicable. It is sightly because the bronze spring wire running along the sole of the shoe blends with the russet leather, while the spiral leggings completely hide the upper part of the splint. They also give added security. It is simple because it is made of two pieces of wire weighing but 2⅓ ounces, and when applied has no point of uncomfortable pressure or of constriction. It is serviceable because it not only holds the foot well extended at all times, but also aids in walking, as it gives the foot the normal flexibility which is lacking in all of these cases.

Pieces of bronzed spring steel wire, 25 inches long, have loops (¾ of an inch long) bent on each end, in opposite directions (Diag. 1). The lower loop is for aid in attaching the wire around the shoe, while that at the top protects the clothing from the sharp wire.

To give the splint flexibility, the wire has a 1¾ turn coil ¾ inch in diameter, bent into it 6 inches from the lower end (Diag. 2). This coil, when in place, is directly opposite the junction of the sole and heel of the shoe.

Two wires, bent as described above, are held in place, one on either side of the shoe, by four pieces of fine piano wire; one piece under the instep, another around the heel at the junction of the upper and heel; a third under the sole, connecting the lower extremities of the wire; and lastly, a piece around the toe between the sole and the upper. The last piece binds the splint permanently to the shoe. It is best to apply the wires in the above order (Diag. 3).

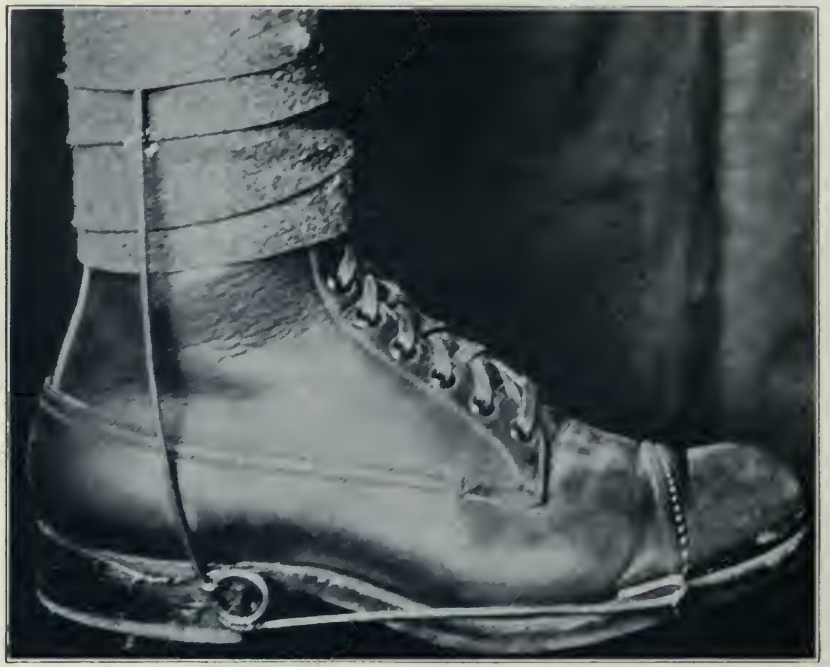

Fig. 1.—Splint for foot drop made of No. 11 spring steel wire and held to the shoe with four pieces of piano wire. The splint is held to the leg by a canvas band passing around the calf between the two wires.

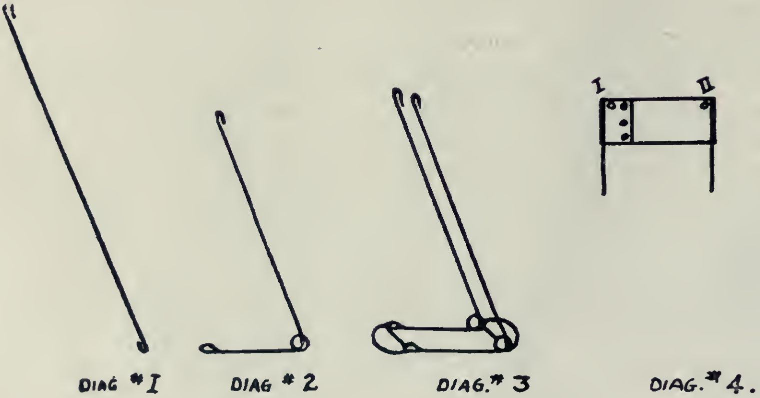

Fig. 2.—Details of splint for foot drop.

The splint is completed by a piece of double ply canvas, 6 inches wide and 4 inches long, and held together by five rivets placed as shown in Diagram 4. Rivets I and II keep the splint from slipping through the canvas. The canvas is slipped over the upper end of the splint, around the calf, and the whole is held in place with a spiral legging.

It takes thirty minutes to make and attach the splint, and it lasts from two to three months.

SPLINT FOR CASES OF WRIST DROP

A wrist drop splint, which is 100 per cent. effective, must perform five definite and distinct functions. First, it must extend the hand on the wrist. Second, it must extend the proximal phalanges on the metacarpal bones. Theoretically, all of the phalanges should be extended, but by actual measurement the small amount of relaxation thus obtained was decided to be less important than the maintenance of normal mobility of the distal joints by permitting free movement. Third, it must extend the thumb, and by extension is meant a position in a plane with the other fingers. Fourth, it should maintain the palmar arch. Finally, it should be light, simple and comfortable, and should give the part as much freedom of motion as is desirable.

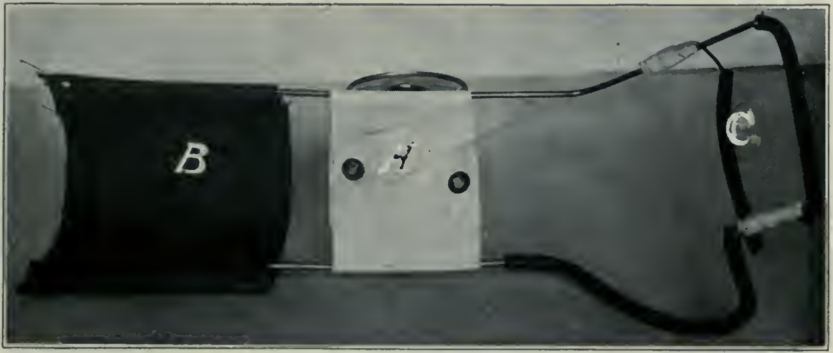

Fig. 3.—Splint for wrist drop with extra piece to support palmar arch. A, adhesive band which fits on dorsal surface of wrist; B, removable canvas band which fits on anterior surface of forearm; C, palmar piece to maintain palmar arch held to the main splint by adhesive plaster.

The old aphorism that multiplicity of methods proves lack of a satisfactory one, certainly holds true for the splints used in cases of wrist drop. In the past few months no less than sixteen different types of splint, from home and abroad, have found their way into this hospital. According to our standards, none of them have been even theoretically 100 per cent. efficient, and but few attain over 40 per cent.

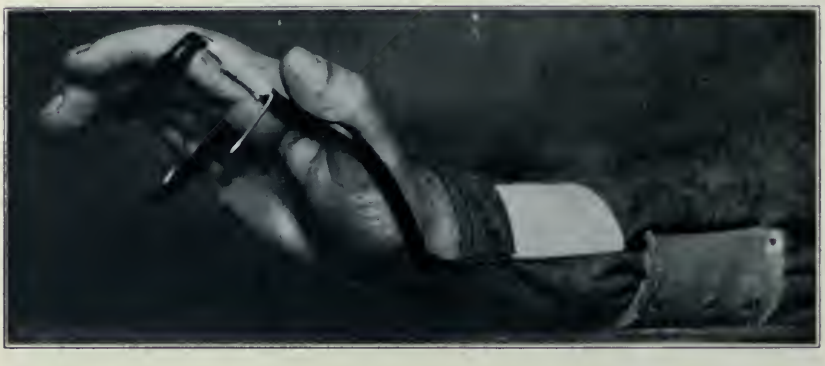

Fig. 4.—Wrist splint drop applied. Thumb is held extended in a plane with the rest of the fingers; distal two phalanges are left free so as to permit normal movement. No bandages are necessary to hold the splint in place.

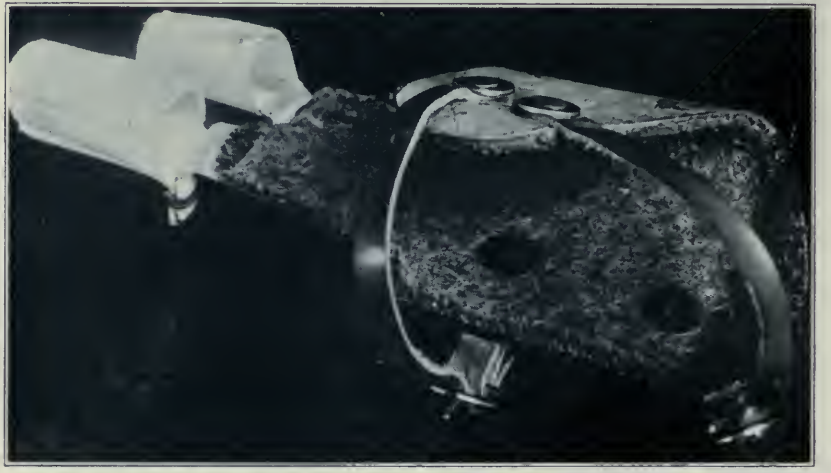

Fig. 5.—Splint for ulnar paralysis. It is made of galvanized iron, padded with felt. The felt is stuck to the iron with sealing wax. Four copper rivets hold the straps in place. The whole is then fitted to the ulnar side of the hand. A forward bend of the finger extensions corrects the tendency to hyperextension. The finger pieces made of galvanized iron padded and surrounded with adhesive tape are held to the main splint with rubber bands which produce the required extension.

Spring steel wire covered with rubber tubing at all points of pressure and properly bent has been found to make a light and comfortable splint. It will meet the requirements and overcome some of the objections of other types. This splint is made from a piece of No. 11 spring steel wire, 35 inches long.

It is best to start bending the splint on the ulnar side of the forearm. Nine inches from the end, the wire is bent upward to form an angle of 140 degrees. This is the angle at which the hand is extended on the wrist. From this point the wire extends forward to the junction of the proximal and middle phalanges of the little finger, where it is bent at a right angle in the same plane. Three quarters of an inch from here it again receives a right angle bend, but this time in a plane at right angles to the previous one. This bend causes the wire to run across the palmar surface of the fingers and thereby extends the proximal phalanges on the metacarpal bones. A right angle bend in the same plane, at the outer side of the first finger, and three quarters of an inch farther on another one in a plane at right angles to it completes that part of the splint which keeps the hand in place. The wire then runs backward along the outer side of the first finger until it reaches a point opposite the base of the distal phalanx of the thumb, where it is bent outward at right angles to extend the thumb on the first metacarpal bone. When it reaches the outer side of the thumb the wire is curved downward and inward to the wrist, following the

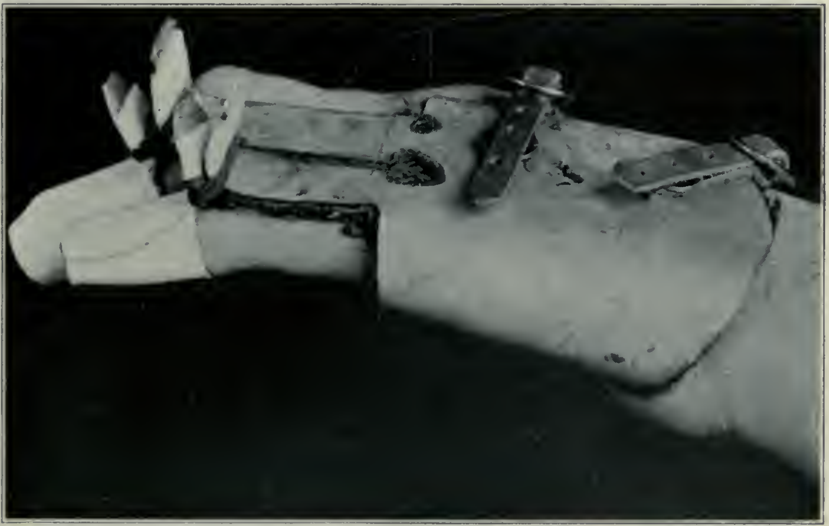

Fig. 6.—Splint for ulnar paralysis applied.

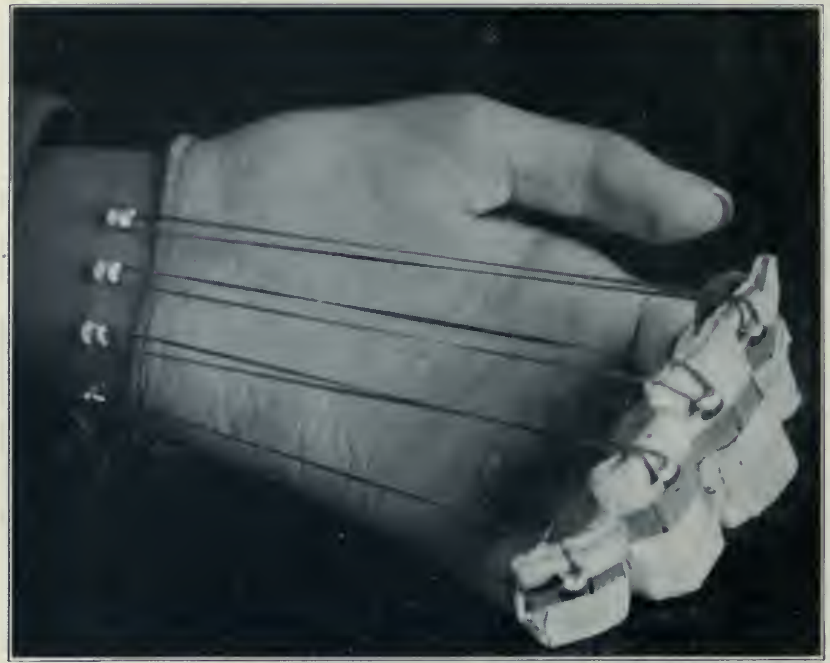

Fig. 7.—First position of twelve hour splints for cases of fixation of the fingers. The splint is worn in this position for twelve hours out of the twenty-four.

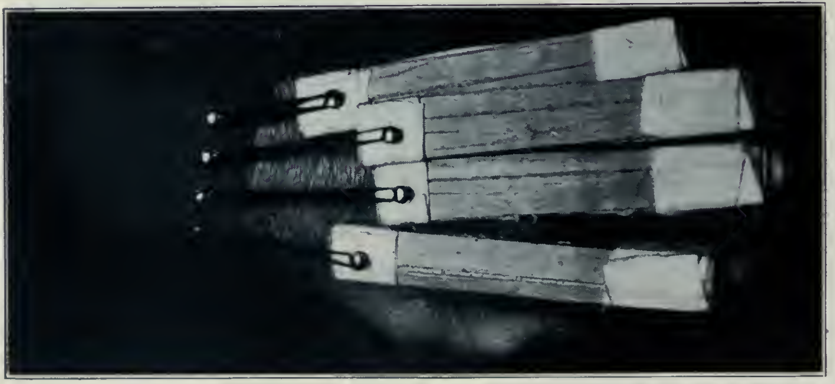

Fig. 8.—Second position of twelve hour splints.

thumb as nearly as possible in its extended position. The wire is now bent so that it runs parallel to and in the same plane as the wire on the ulnar side of the forearm. The two rubber tubes which protect both the fingers and the thumb from the splint are now slipped into place, and then the ends of the wire are bent on themselves, thus protecting the patient from the sharp ends. The palmar arch support is made from No. 20 spring steel wire, covered with rubber tubing, and is attached to the splint by adhesive plaster. The ends are one inch long and are bent in opposite directions from each other to increase the stability of the palmar attachment. The wire first extends downward away from the splint for an inch on each side and then by a slow curve extends upward to support the palmar arch. A piece of adhesive plaster is now placed on the splint just above the wrist and folded on itself so that all of the sticking surface is covered. Finally, the canvas band is slipped over the ends coming in contact with the flexor surface of the forearm.

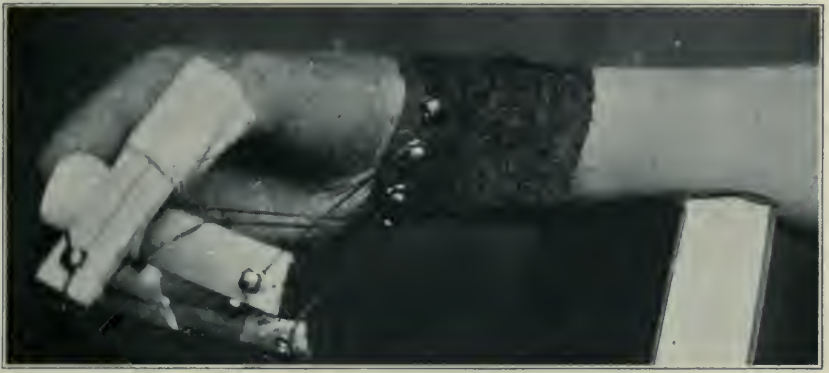

Fig. 9.—Splint for median paralysis applied. Finger pieces are made of thin board held together by strips of adhesive tape.

This splint can be made in fifteen minutes. It is applied without bandages and can be readily removed or put on by the patient, without assistance.

SPLINT FOR ULNAR PARALYSIS

A very satisfactory ulnar splint, which not only aids in extending the fingers but also allows them free motion, is made from a piece of galvanized iron cut from a pattern, but shaped to the hand of each individual so that it closely grasps the ulnar half of the hand. It is held in place by two straps, one just below the hypothenar eminence and the other just above the web between the thumb and the first finger. To give additional comfort, the splint is lined with blanket material stuck to the galvanized iron with sealing wax. The forward projections are of such length that when one half inch is turned up the projections extend just beyond the first phalangeal joint. Another piece of galvanized iron one half inch wide and one and one half inches long is bent at right angles so that one half inch of it corresponds to the half inch upward bend on the projection of the main splint. Around the remaining one inch adhesive tape is wrapped so as to form a cap for the end of the finger, the inner part of this cap being lined so that it can be removed or applied at will. A rubber band binding the two upright extensions together completes the splint.

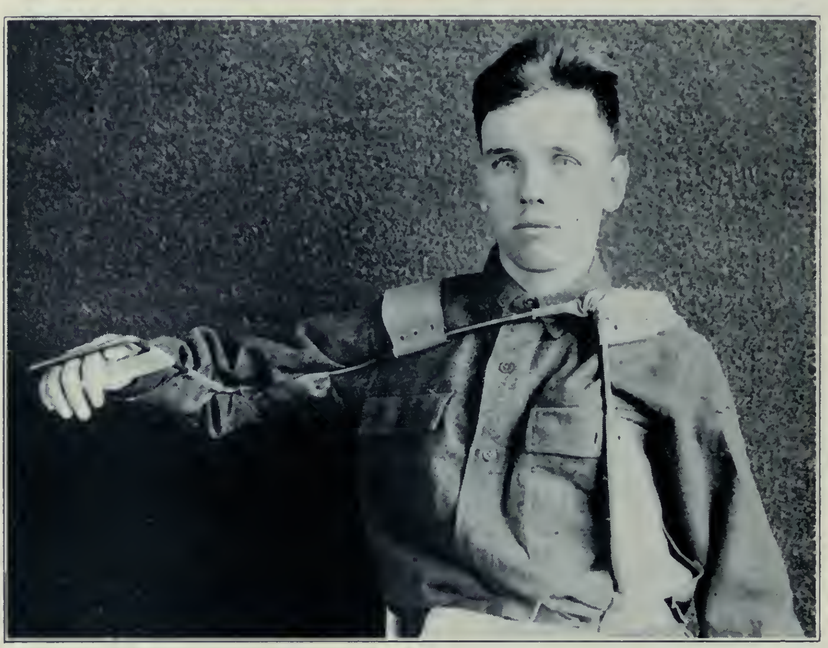

Fig. 10.—Splint for brachial plexus cases applied. It transfers the weight to the opposite shoulder and to the thigh of the same side. The splint proper is made from $\frac{3}{16}$ iron. The canvas straps are held in place with eyelets.

TWELVE HOUR SPLINT FOR BRACHIAL PLEXUS CASES IN WHICH THE FINGERS HAVE STIFFENED IN SEMIFLEXION

Wooden coaptation splint material is used. Short strips are cut, the ends being protected by adhesive plaster. Rings of adhesive plaster, lined so that they do not stick to the skin, are stuck to one end of this splint and fitted to the fingers. A shoe hook is set in the other end of the splint. The shoe hook is then fastened by a rubber band to a corresponding shoe hook placed in a canvas wrist band.

The patient is supplied with a long and a short set of these splints, and is told to wear them interchangeably — the short set to produce flexion and the longer ones to produce extension.

SPLINT FOR MEDIAN NERVE CASES

The splint for the median nerve case (inability to flex the thumb and fingers) differs in no way from the flexion service used in the twelve hour splint, except that it includes the thumb.

SPLINT FOR CIRCUMFLEX NERVE CASES

This splint was copied from one sent from the Lakewood Hospital. It is made from $^3/_{16}$ inch iron, and is much lighter and very much less disagreeable to wear than the old aeroplane splint formerly used for these cases. The points of pressure in this splint are the sound shoulder and the hip of the affected side.

410 Overland Building.

A CASE OF MENINGO-ENCEPHALITIS (LETHARGIC ENCEPHALITIS)*

WILLIAM W. HALA, M.D. AND CYRIL M. SMITH, M.D.

BROOKLYN

The purpose of this article is to report a case, clinically diagnosed as encephalitis lethargica, verified by observations antemortem and postmortem.

As a result of the influenza epidemic which raged in Southern and Middle Europe in 1890, cases of epidemic lethargy appeared in Italy and in Hungary.[1] Economo,[2] writing about an epidemic which occurred in Vienna in 1917, likewise speaks of these cases, which at the time were called "nona." He introduced the term encephalitis lethargica. Wilson[3] gives a description of the pathologic changes in the brain of cases of this disease and ascribes the cause to a toxi-infective origin.

Von Weissner, as mentioned in the special article on lethargic encephalitis in *The Journal of the American Medical Association,* March 15, 1919, recovered a gram-positive diplostreptococcus which he claims produced somnolence when injected into apes. Up to the present time, however, no organism has been described which could be held as the etiologic cause of this disease.

Among the recent literature in this country, O. L. Pothier[4] made a report of eight cases occurring at Camp Lee with necropsy findings in one case. Bossoe[5] of Chicago then reported a series of twelve cases observed by him, with two necropsies. The histologic changes that he observed are very definite and agree substantially with those observed in our case. Finally, Tucker,[6] in a recent article, calls attention to eleven cases, with necropsy findings in two of them.

The case reported in this paper is interesting because it displayed not only the characteristc histopathologic lesions observed in the brain by others, but also on account of the intense purulent meningitis and ependymitis found at necropsy. The fact that an organism was isolated both antemortem and postmortem, which evidently was the etiologic factor concerned, is noteworthy.

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* From the pathological Laboratory, Kings County Hospital.

[1] Netter: Lethargic Encephalitis, Paris méd. **8**:81 (Aug. 3) 1918; Bull. de l'Acad. de méd., Par. **79**:337 (May 7) 1918.

[2] Economo: Verein f. Psychiat. u. Neur. in Wien, April 17, 1917.

[3] Wilson, S. A. K.: Lethargic Encephalitis, Lancet **2**:7 (July 6) 1918.

[4] Pothier, O. L.: Lethargic Encephalitis, J. A. M. A. **72**:715 (March 8) 1919.

[5] Bossoe, P.: Epidemic Encephalitis, J. A. M. A. **72**:971 (April 5) 1919.

[6] Tucker, B. R.: Epidemic Encephalitis Lethargica, J. A. M. A. **72**:1448 (May 17) 1919.

Vaughan,[7] in an excellent résumé, has discussed the disease from a clinicopathologic standpoint in a recent issue of the *Journal of Laboratory and Clinical Medicine.*

Dr. John H. Larkin[8] has informed us of two cases of hemorrhagic myelomalacia that he has observed both clinically and at necropsy, in which the histopathologic changes in the cord were similar to those present in the brain of our case. These cases are to be reported shortly.

REPORT OF CASE

History.—Our case is that of a male, aged 31, a bottler, who was admitted to the general medical service of Drs. Hall and Merrill in Kings County Hospital, Brooklyn, March 24, 1919. The family history was negative except that his wife had died of influenza two months previously. His first visit to a physician was on March 14, at which time his ailment was diagnosed as influenza. The attack was not sufficiently severe to confine him to bed continuously. Two days prior to admission he collapsed suddenly, and from that time he appeared somewhat drowsy.

His chief complaint on admission was malaise, headache and fever. He appeared dull and drowsy, and his responses to questions were slow and retarded. General physical examination was negative except that the neck and back were somewhat rigid, yet the neck could be turned without manifestations of pain. Kernig's sign was absent, and other reflexes, deep and superficial, were not exaggerated. Tactile sensation was present.

Symptomatology.—Examination made on the day following admission showed increasing rigidity of the back and cervical region, but not sufficiently severe to produce opisthotonos. No pain was evinced on active rotation of the neck, but limitation of movement of the head on the shoulder, most marked on the right side, was present. The pupils were dilated and sluggish. His face assumed a mask-like appearance, and he responded to questions though seemingly not understanding. He was not excitable. Superficial and deep reflexes were present and apparently normal; no tâche cérébrale. He swallowed liquids without difficulty.

On March 27 the patient's condition was slightly improved, but he maintained the drowsy appearance and listless attitude. The eyes made feeble attempts to follow moving objects. At times respiration was noisy but never labored. Eye disturbances were negative except for a mucopurulent discharge from the palpebral conjunctiva. A slight bilateral ptosis was present, evidencing a disturbance of the nerve supply in the muscularis palpebrarum. The tongue was dry and parched, and the face muscles on the left were apparently weakened allowing the mouth to deviate to the right.

On March 28 the pupils reacted to light and accommodation, the right being slightly irregular in contour. Efforts to arouse him were futile, but he would make an attempt to protrude his tongue after repeated commands, and he would try to grip hands. Examination of the superficial and deep reflexes showed a sluggish right abdominal reflex; right achilles present and sluggish; no Babinski reflex, Chaddock's, Oppenheim's or Westphal's sign.

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[7] Vaughan: Encephalitis Lethargica, J. Lab. & Clin. M. **4**:381 (April) 1919.

[8] Larkin, J. H., Director Strecker Memorial Laboratory, City Hospital, New York. Oral communication.

On March 29 both pupils reacted similarly to the day previously, though not so sluggish. The ptosis was more pronounced. Urinary incontinence developed.

On March 30 the deep and superficial reflexes had entirely disappeared; the body was more rigid. The pupils were irregular in contour and immobile. Horizontal nystagmoid movements were pronounced, and there was evidence of a partial paralysis of the left external rectus. The disks were edematous, but not atrophied. The retinal vessels were congested and tortuous. On account of urinary retention, catheterization was necessary, and obstinate constipation yielded to enemas. Feeding by stomach tube was instituted because of the increasing dysphagia. Toward night increased difficulty in breathing developed, associated with a tendency to pulmonary edema. The semicomatose condition of the previous days had assumed a lethargic character. The tongue deviated to the right. There was increased paralysis of the left facial muscles with a jaw clonus, rapid and regular in rate.

On March 31 the rigidity of the body had entirely disappeared. The pupils were small and immobile. Complete paralysis of the left external rectus of the eye and of the muscles of deglutition were evident. Toward evening pulmonary edema increased, and the patient died at midnight.

Clinical Course.—On admission, his temperature was 104 F.; pulse 96, regular and of good volume; respiration 28. During the next three days the temperature varied from 101 to 103.2, reaching 105 at 8 a. m., March 29. From that time until death the temperature maintained a fairly normal level at 104 F. There does not appear to be any relation between the pulse rate and the temperature (Fig. 1).

Laboratory Findings.—A cytologic examination of the spinal fluid the first day showed a total of 480 cells per c.mm., with a differential count of eighty-four polymorphonuclears, and sixteen lymphocytes; Noguchi globulin test +; no bacteria found in smear; culture negative. Subsequent cytologic findings on the 28th, 400 cells, 88 per cent. polys, 12 per cent. lymphs, globulin +; culture, negative; smear, negative.

Wassermann test negative on blood and spinal fluid. Lange's test on two occasions was as follows: Before administration of 40 c.c. Flexner's antimeningococcic serum 0 0 0 0 1 3 2 1 0; after serum 0 0 0 0 1 1 1 0 0.

Urine, the specific gravity was 1.024, amber, acid, no sugar, trace of albumin, 3.5 per cent. urea, 1.2 per cent. creatinin, trace of mucus, hyaline casts, few epithelial and white blood cells.

Widal test on 29th with 1:20 dilution was negative, blood urea 34 mgm. per 100 c.c., blood sugar, 0.12 per cent.

Spinal punctures were done with the following results: 25th, 2 c.c., negative; 26th, fluid clear, increased pressure, no débris or cloudiness; 27th, dry tap; 28th, no change in character; 29th, no change in character, culture again negative and smear negative; 30th, fluid slightly cloudy, under pressure; cell count 12,000 per c.cm., 90 per cent. polys, 10 per cent. lymphs. Examination of smear showed a motile bacillus, gram-negative, staining readily with ordinary anilin dyes.

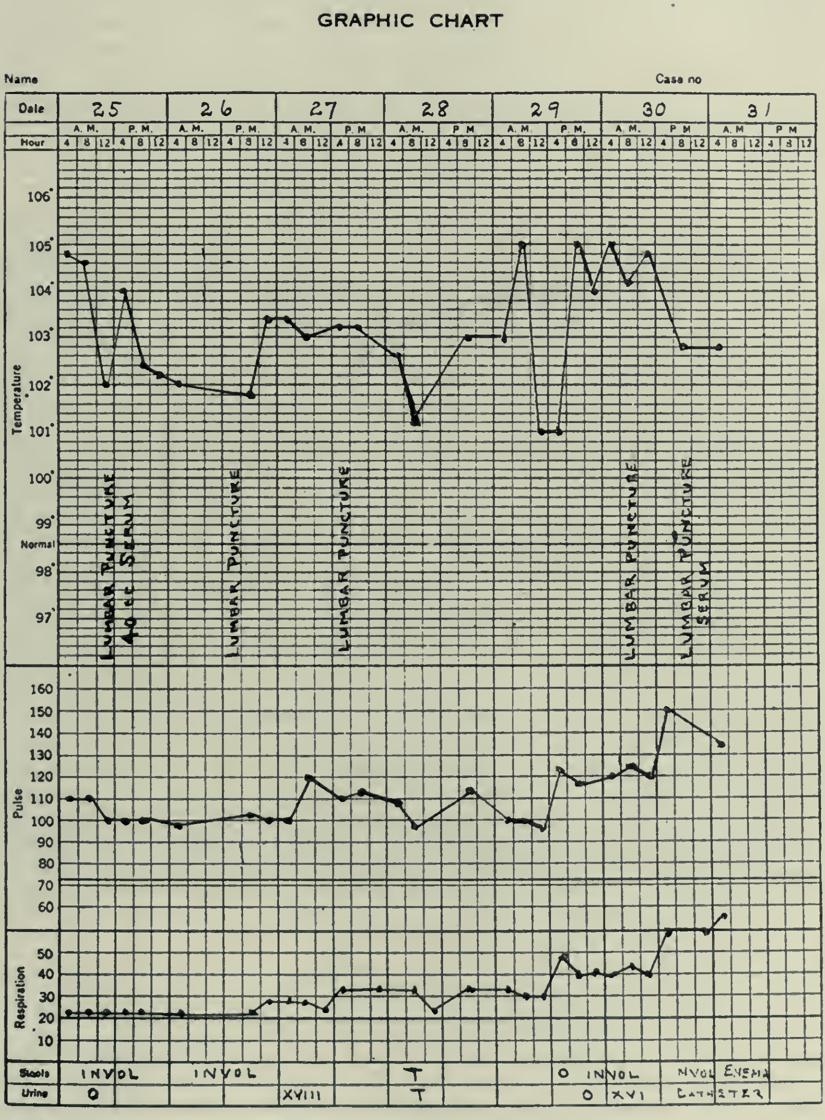

Fig. 1.—Temperature chart.

Differential and total white blood count: March 27, 12,000, 76 per cent. polys, 24 per cent. lymphs; March 21, 24,000, 85 per cent. polys, 15 per cent. lymphs.

Necropsy (performed April 1, 1919).—Necropsy on the body shows the following: Congestion and edema and lobular pneumonia of lungs; septic spleen; parenchymatous degeneration of the liver; acute nephrosis of left kidney; cystic remains of right kidney with anomaly of ureter; subdural hemorrhage; acute exudative meningitis of brain and cord; acute encephalitis; acute ependymitis; petechial hemorrhage in corpus striatum, centrum ovale and optic thalamus, right side.

Head: On removing the calvarium we found the dura adherent over the entire right hemisphere. The brain was therefore removed with the dura attached. On reflecting back the dura of the left hemisphere, the entire cerebral cortex was injected, the pial vessels being rather prominent in places. Small hemorrhagic areas were seen in the pia, and in the sulci between the convolutions a purulent exudate was seen, especially along the fissure of Rolando. On the right side the adherent dura apparently incorporated an oval, rather firm tumor, compressing the motor area of the right cerebral cortex. Incision into this dural tumor disclosed an encapsulated mass, brownish red in color and soft in consistency, suggestive of subdural bloodclot. Observation of the base of the brain, particularly of the pia of the cerebellum, pons and surrounding surfaces of the temporal lobes and of the crura cerebri, likewise revealed marked injection of the vessels and the presence of a yellowish purulent exudate. Cultures of the exudate were made under careful aseptic precautions. The brain was then placed in fixing fluid for later study.

Cord: On removal of the cord and opening up of the dural cavity, the pia was injected and covered, in the cervical and lumbar regions, by a yellowish purulent exudate. Cultures were made from this exudate.

Brain: After the brain had been sufficiently hardened it was sectioned anteroposteriorly by direct cross section beginning at the anterior portions of the cerebral hemispheres and continuing, by parallel section, to the cerebellum. In the right centrum ovale and at the region of the first portion of the anterior horn of the right ventricle petechial hemorrhages were present in the roof and lateral wall of the ventricle. This petechial hemorrhagic condition was still present, especially in the head of the corpus striatum on the right side. The optic thalamus of the right side showed punctate hemorrhages. The ventricle, including the anterior and middle horns, was filled with a purulent exudate. There was apparently no invasion of the hemorrhagic infiltration into the lenticular nucleus or the internal capsule or into the claustrum on the right side. The right ventricle in the region of the posterior horn appeared to be free from purulent exudate. The left ventricle in the anterior portion of the middle horn and in the entire posterior horn showed purulent exudate. The basal ganglions on the left side were apparently free of petechial hemorrhagic areas. The purulent exudate was marked on the pia covering the basal ganglions, particularly in the recesses of the crura cerebri. The portion of the white matter and basal ganglions affected by the punctate hemorrhages showed undue softening.

Microscopic Findings: The most important and relevant changes were found in the brain and cord. The meninges of both brain and cord showed a purulent exudate. The bacillus isolated by culture, both antemortem and postmortem, was found present in this exudate both by Gram-Weigert and thionin stains:

"pari passu" we have used the thionin stain in cases requiring the demonstration of organisms in the tissue as a control on the Gram-Weigert stain, particularly in cases of gram-negative bacteria.

Sections showing the roof of the ventricle, including a portion of the centrum ovale, exhibited petechial hemorrhages and perivascular small round cell infiltration. The ependyma of this ventricle was infiltrated by pus cells among which the bacillus was found by Gram-Weigert and thionin stains. The sections of the head of the right corpus striatum showed distinct inflammation with petechial hemorrhages and perivascular infiltration. Sections of the pons and medulla were normal, except for the presence of the purulent exudate on the pia. Cross sections of the crura cerebri taken in the region of the origin of the third nerve showed perivascular round cell infiltration but no petechial hemorrhages. Where the nerve emerged the pia showed considerable purulent exudate.

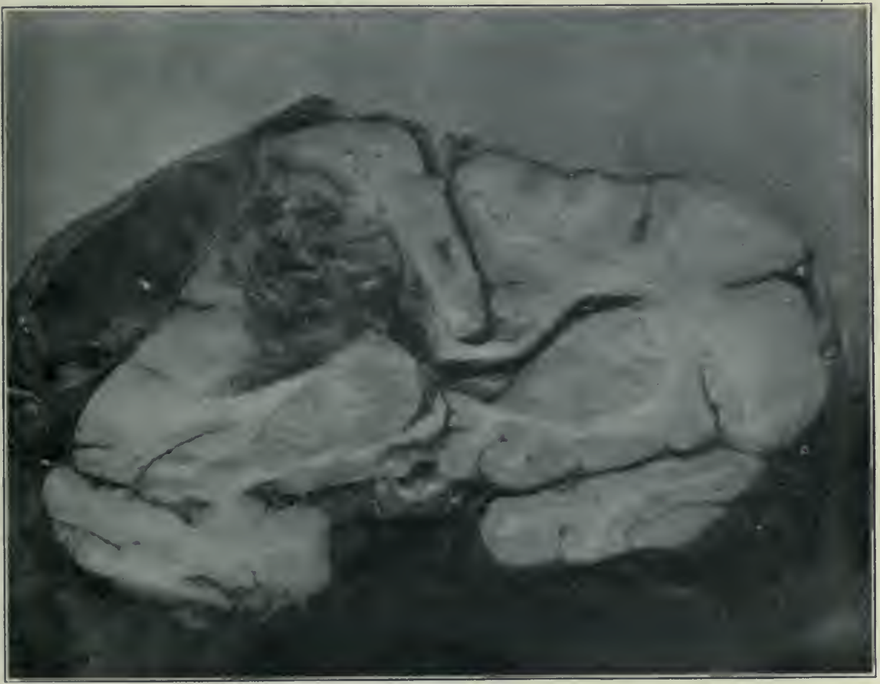

Fig. 2.—Brain, showing lesions grossly.

Sections of the subdural hemorrhagic tumor all showed an extension of the meningitic exudate into the bloodclot.

Cross sections of the cord at different levels showed a pure septic meningitis. The organism was again demonstrated by stain.

Bacteriologic Data: On March 30, 1919, one day before the death of the patient, a specimen of spinal fluid was received at the laboratory. It showed 12,000 cells of which 90 per cent. were polynuclears and 10 per cent. mononuclears, with globulin in excess. A stained smear showed the presence of a small gram-negative motile coccoid bacillus. The fluid was cultured, and

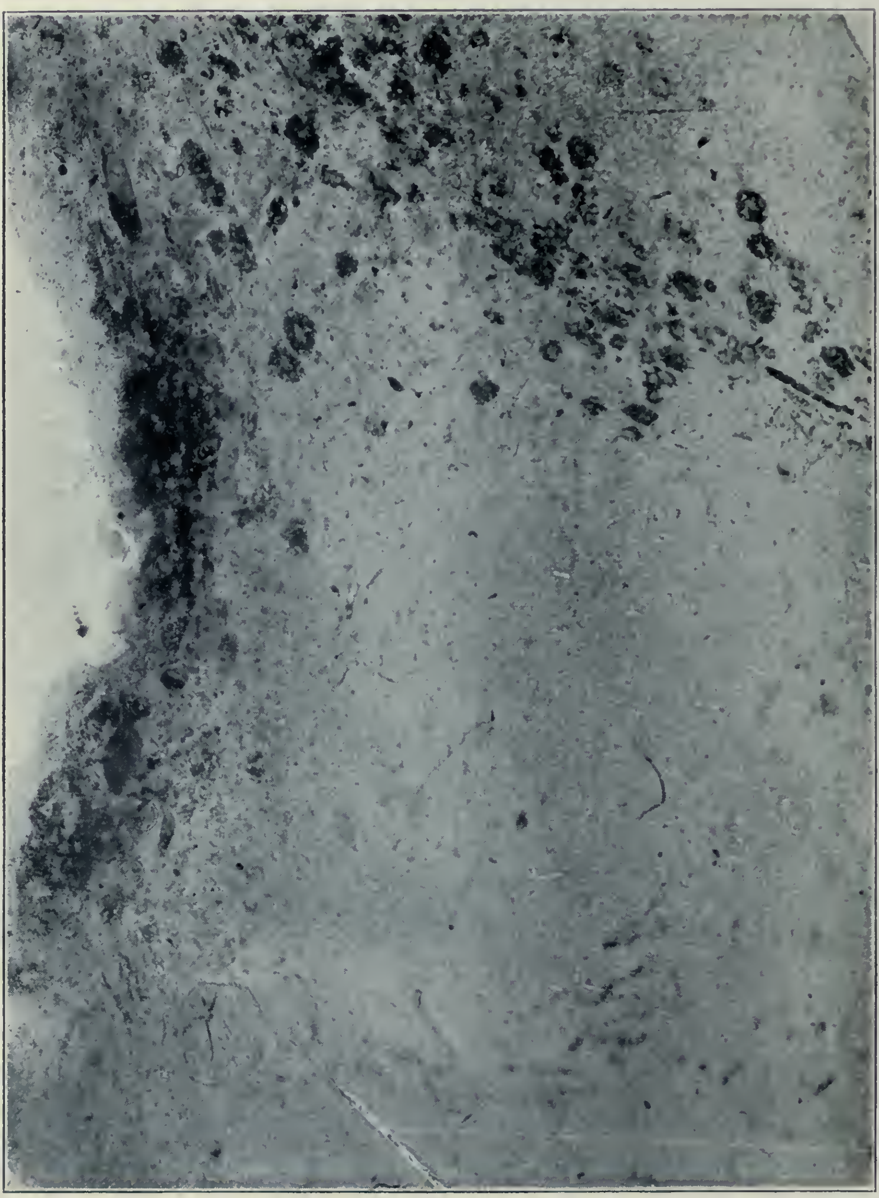

Fig. 3.—Section showing centrum ovale and ependyma of ventricle. Note petechial hemorrhages in the substance of the centrum ovale and acute purulent ependymitis.

a pure growth of this bacillus was obtained on both plain and blood-enriched mediums. In size it was 0.8 micron by 0.3 micron. In subsequent cultures the size varied greatly, some of the bacilli approaching 1.2 microns in length and some suggesting polar bodies.

Inoculations were made into a guinea-pig, a mouse and a rabbit. These animals, after a lapse of from about three to seven days, died. While sick none of them showed any stupor. Necropsy showed purulent peritonitis only, all inoculations having been intraperitoneal. The organism was recovered in pure culture from the abdominal cavity of each animal. The rest of the organs of the animals were normal, including brain and cord.

On April 1, 1919, at the time of necropsy on the body of J. K., cultures were made from the cerebral cortex and the purulent exudate on the base of the brain and the exudate on the cord. All of these cultures showed the same organism as that found in the antemortem cultures of the spinal fluid. An intravenous injection of 0.5 c.c. of bouillon culture was made into a rabbit. This injection was made April 7, the animal dying April 11. No evidence of stupor was observed while the animal was alive, although it appeared quite sick and disinclined to move about. Necropsy made on this rabbit revealed no gross lesions in the organs; and when we studied them, none of the animals inoculated showed any gross or microscopic evidence of infection of the brain or cord. The organism was recovered in pure culture from this rabbit's heart's blood.

Two guinea-pigs were injected intraperitoneally with cultures from the cerebral cortex: one lived seven days, the other three days. Necropsy showed purulent peritonitis. The organism was recovered from both animals in pure culture. We are still unable to classify the organism. We tested the original cultures obtained antemortem from the patient's spinal fluid, postmortem cultures and cultures from the inoculated animals. All grew on the chosen mediums, and all reacted alike on the sugars. No characteristic growth was observed on any of the mediums which would aid us in differentiating it from other organisms of like morphology. On sugars the following reactions were noted:

Glucose	Acid—Gas
Lactose	No change
Saccharose	No change
Mannite	Acid—no gas
Inulin	No change
Dextrin	No change
Maltose	No change
Galactose	Acid—no gas
Levulose	Acid—no gas
Litmus milk	No change

No indol production

Agglutination test with typhoid and paratyphoid A and B serums, negative.

The organism grew best aerobically. Its motility was quite marked in fresh cultures. In older cultures its motility was constantly lessened.

The cultural characteristics of this organism on sugars coincide with those of no other organism of which we know. Its action on the sugar mediums was tested several times and agreed with the original findings each time. There is no doubt that this organism was the cause of the patient's meningitis; and in

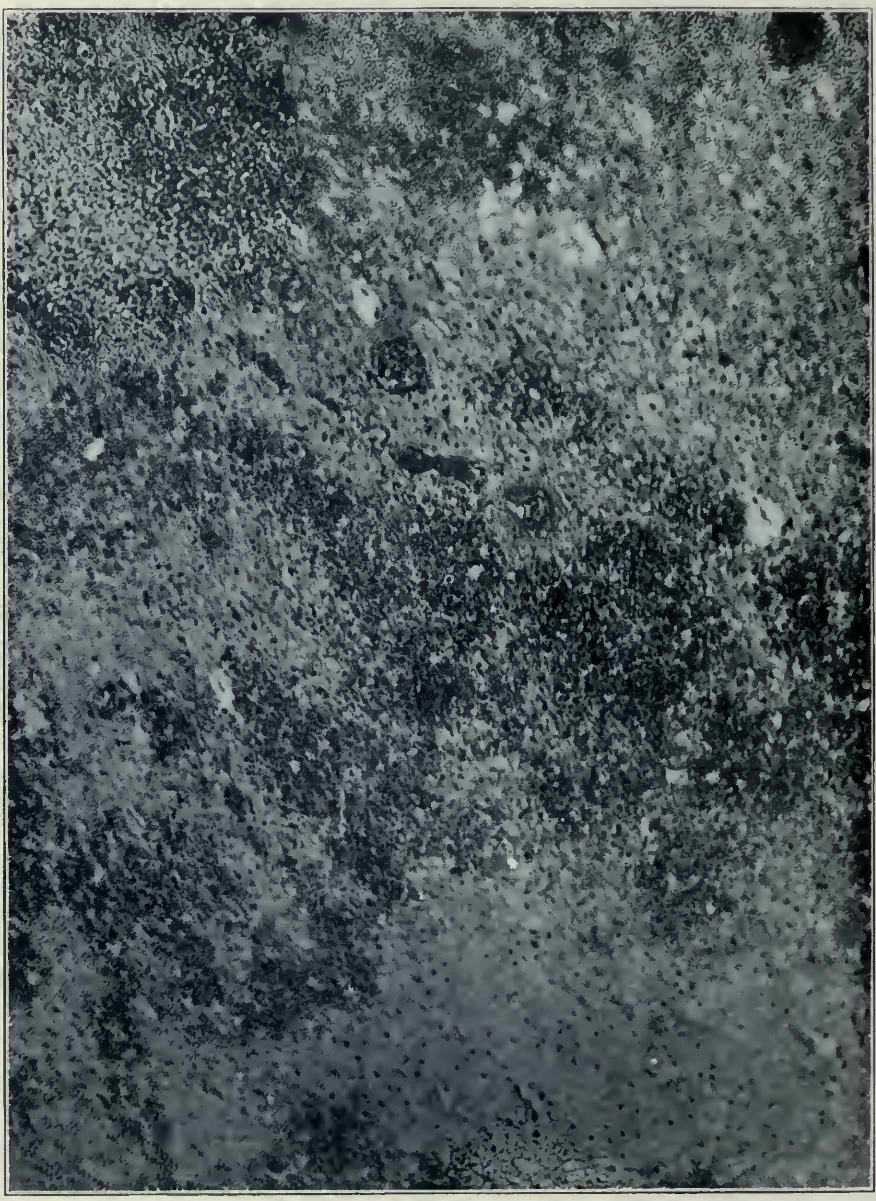

Fig. 4.—Section of corpus striatum; marked hemorrhagic extravasation, thrombotic vessels and exudative inflammation.

view of the fact that it was demonstrated in the purulent exudates of the ependyma of the ventricles, we are of the opinion that it probably caused the inflammatory changes and petechial hemorrhages of the centrum ovale, corpus striatum and the optic thalami.

SUMMARY

From the clinical standpoint our case was one of meningo-encephalitis, with lethargy and involvement of the motor fibers of the third, sixth, seventh, tenth and twelfth cranial nerves.

The etiologic cause was a gram-negative motile bacillus, unidentified, but probably belonging to some intermediate class of the colon-typhoid-enteritidis group.

Pathologically, the lesion demonstrated septic meningo-encephalitis and ependymitis, with punctate hemorrhages and perivascular cell infiltration of the centrum ovale, corpus striatum and optic thalamus.

We are indebted to Drs. Nidish and Atchley of the laboratory staff, and to Dr. Peter Lavelle of the intern staff, for their cooperation and the work which they did on this case and to suggestions and aid given by Dr. John H. Larkin of Strecker Memorial Laboratory, City Hospital. For permission to publish this case we wish to express our thanks to Drs. Hall and Merrill, on whose service the case occurred, and to Dr. C. F. Barber, president of the Medical Board of Kings County Hospital.

Abstracts from Current Literature

TRAITEMENT INTRARACHIDIEN DES AFFECTIONS SYPHILITIQUES ET PARASYPHILITIQUES DU SYSTEME NERVEUX.

Gonzalo R. Lafore, Rev. neurol. **26**:625 (Aug.) 1919.

The author reviews the works of Nonne, Plaut and others, showing that general paralysis and tabes are of syphilitic origin; Noguchi and Moore's demonstration of the spirochete in the cerebral cortex of paretics and in the cord of tabetics; Noguchi's proof of spirochetes in parasyphilitics, and the work of Nichols, Houghman and others who demonstrated the existence of the spirochete in the spinal fluid of monkeys and other animals after infecting them through the testicles and the conjunctivae.

He then raises the question of why treatment has not been efficacious in the so-called parasyphilitics (paretics and tabetics). He reviews the discussion of Goldman, Weed, Frazer, Dickson, Halliburton and others on the subject of the origin and the movement of the spinal fluid. He does not accept the idea of Mott, Dickson and Halliburton that the fluid passes from the subarachnoid space through the capillaries, etc., as he thinks that the greater pressure is on the side of the blood stream, pressure in the veins being less than that of the cerebrospinal fluid. Toxic substances secreted in the brain by the spirochete pass into the perivascular lymph current; from the cranium they pass to the spinal canal.

There is, however, a current, he thinks, moving in the opposite direction (as has been demonstrated in the case of tetany treated by the method of Doyen). These two counter currents converge in the direction of the bulb.

By animal experimentation the author has produced through spinal injection a diffusion of coloring matter through the cord, bulb, base, encephalon and the olfactory nerve, as well as by penetration through the ependymal canal. If the animal lives long enough the convexity of the brain and the surfaces of the longitudinal fissures also show the coloring matter. Intraventricular injections show coloring matter in the aqueduct of Sylvius, the fourth ventricle, Ammon's horn and the like.

That mercury and arsphenamin have appeared to irritate the cortex is seen in speech difficulties, nausea, headaches, etc. He cites the case of Cotton's patient who had had an intraspinal injection one day, who died the next, and in whose ventricle, arsphenamin was found the following day. The author himself had a case in which he demonstrated that arsphenamin rose from the lumbar region as high as the cerebral region in half an hour.

The author agrees with Leredde on the importance of treating the infection rather than the symptoms and believes that treatments should be pushed as long as laboratory findings are positive, even though clinical manifestations have disappeared. He does not agree with Dejerine who maintains that the mercurial treatment of tabes and general paresis differentiates these from secondary syphilis, but he does agree with Leredde that vigorous intravenous and intraspinal injections bring about important changes in the cerebrospinal fluid in tabes and, though not so much so, in general paresis. He thus quotes Leredde's résumé:

1. The result of the treatment is certain and shows itself in all the changes of fluid.
2. Lessening of lymphocytosis is the rule, and may be quite marked.
3. Hypertension usually disappears.
4. In contrast to the results in general paresis, the findings of the fluid in tabetics yield very strikingly to neo-arsphenamin.
5. Quantitative diminution of albumin and globulin runs parallel with the disappearance of the serologic reaction.

Many of the author's patients who have not yielded to mercurial preparations and to intravenous treatment have improved remarkably under intraspinal treatment. Certain patients call for both intravenous and intraspinal treatment because of an involvement of the vessels, viscera and general body structures as well as of the central nervous system.

The ideal method in the treatment of syphilis of the central nervous system is that which reaches readily the site of the lesion in a direct way and in exact dosage and not in a manner that depends on the varying filtrating qualities of the choroid plexus of the brain or the cord.

The author uses the terms "benign" or "latent" for those cases of syphilis in which the patient never has known that he had syphilis and for those cases in which the patients have had a chancre which was considered nonsyphilitic and which disappeared following local treatment.

Laredde finds that 50 per cent. of his female cases and 28 per cent. of his male cases fall under this classification; 40 per cent. of the author's tabetics and 35 per cent. of his general paretics also fall under this classification.

The laboratory findings frequently show syphilis of the central nervous system before any clinical symptoms appear and so permit diagnosis of the early stages of general paresis which frequently is looked on as neurasthenia. It is these patients that are benefited most by treatment

The Diagnosis of General Paresis and Cerebrospinal Syphilis.—The author then enumerates the five laboratory signs and reactions for the differential diagnosis of general paresis and cerebrospinal syphilis: (1) Wassermann reaction on the blood; (2) increase of globulins; (3) lymphocytosis and presence of plasma cells; (4) Wassermann reaction on fluid, and (5) Lange's colloidal gold test.

The first three of these reactions may be present, either in paresis or in cerebrospinal syphilis but the fourth and fifth may not appear. The author states that from 80 to 95 per cent. of all patients show a positive Wassermann reaction on the spinal fluid if not more than 0.2 c.c. of fluid are used, but the reaction is usually negative in syphilis and tabes. Only 20 per cent. of tabetics show a positive Wassermann reaction on the fluid. Those patients with syphilis of the central nervous system in whom there is a negative fluid reaction may show a positive fluid reaction on using from 0.3 to 1 c.c. of fluid.

The author then raises the question whether it is possible, by the aid of the Lange reaction, always to differentiate general paresis from other forms of neurosyphilis. He thinks that one may be reasonably sure of the diagnosis of paresis if it has the usual clinical symptoms together with the five laboratory reactions, but he believes there are certain cases of the so-called pseudo-general paresis which do not show all the laboratory signs but present the clinical symptoms, and yet at necropsy and by histopathologic study fail to show microscopic changes characteristic of general paresis as established by Nissl and Alzheimer. He then quotes Southard and Salomon's belief that in doubtful cases the patient ought to have the benefit of modern treatment because there are cases in which it is impossible to determine whether the patients are paretics or other types of neurosyphilitics.

The author has seen patients in his clinics regarded as paretic without presenting any characteristic mental symptoms and running a course of ordinary neurosyphilis, and also patients with tabes with optic atrophy who have given laboratory reactions almost identical with those of general paresis. One of his patients had a gummatous lesion involving almost the entire hemisphere; another showed multiple gummas and yet the diagnosis could not be made until necropsy.

Suggestions for Treatment.—The author offers these suggestions for treatment:

1. In the treatment of cases of simple neurosyphilis as judged by the Lange curve, but with positive blood and negative fluid reaction and with an increase of globulins and cells, use may be made of the intravenous method and, if after four or five injections, improvement is not evident, alternate intravenous and intravenous and intraspinal treatment.

2. In the treatment of cases of simple neurosyphilis with both the blood and fluid reaction positive, one should begin with the combined intravenous and intraspinal treatment.

3. In the treatment of cases of simple neurosyphilis with negative blood and positive fluid one should begin with intravenous treatment; Wassermann tests should be taken on several bloods and if they continue negative only intraspinal treatment should be used. Almost all these cases have some concomitant vascular or visceral involvement, and the combined method of treatment is the surer.

4. In the treatment of parasyphilis (tabes and general paresis), both the intravenous and intraspinal method should be employed from the beginning.

The author reviews the various opinions on the relative value of intravenous and intraspinal treatment, especially in cases not tabetic or paretic, and the still more debatable question of the so-called cure of general paresis and tabes. He admits that there are a few indubitable cures by the intravenous method alone, but he emphasizes the fact that there are many more patients that have not been benefited by this method alone, but that quickly improve as soon as intraspinal treatment is introduced. He then cites instances of five such cases in his own practice, including a case with beginning optic atrophy and spastic paraparesis, a case of tabes, one of general paresis, one of vascular syphilis with pseudobulbar lesions, and a case of syphilitic meningo-encephalitis with paraplegia. His account shows rather remarkable improvement, but he does not state whether they are selected cases or not.

Preparation of Serum and Technic of Injection.—The author then describes the technic of his collaborator for the preparation of the serum in vitro. It is put in a flask with two openings, after the blood is drawn; the entire outfit is sterilized in an autoclave at 120 C. After the serum is prepared, it is sealed up in ampules containing 4 mg. of mercuric chlorid and 7 mg. of neoarsphenamin and employs such fractions of the contents as he wishes to use in individual cases. If made carefully, the ampules may be used for a period of several months.

The author's technic of intraspinal infection is:

1. Fifteen c.c. of fluid are drawn off for analysis.
2. The serum is mixed in 5 c.c. of the fluid and injected.

3. The author no longer makes preliminary use of stovain and procain as he has learned that their effects disappear before the onset of the pains due to injection. Instead he resorts to the routine use of pantopon and acetyl-salicylic acid, three or four hours after the injection.

4. He prepares the serum according to the method of Byrnes and Ogilvie.

5. At the beginning of a course of treatment he uses 2 mg. of mercuric chlorid and 3 mg. of neoarsphenamin; later he uses as high as 4 mg. of mercuric chlorid and 7 mg. of neoarsphenamin.

6. Before beginning intraspinal treatment, he gives four or five intravenous treatments at intervals of from two to six days, using 1 cg. of mercuric cyanid and 0.3 gm. of neoarsphenamin.

7. He always uses in the first intraspinal infections arsphenaminized serum, never more than 2 to 3 mg. He has had no severe reactions.

8. In the interval (from twenty to forty days) between intraspinal injections, intravenous injections are employed but never more than two a week, and he always alternates the mercurialized and arsphenaminized serum.

Dosage Employed in Intraspinal Treatment. — Originally, the author gave large dosages producing strong reactions, maintaining such dosages up to the point of complete remission. Recently, however, he has begun his treatment with small dosages, never using enough to produce intense reaction, gaging the interval by the degree of amelioration of symptoms. Tabetics with marked crisis, paretics with seizures, and parasyphilitics with spasticity, should not receive over 2 mg. of mercuric chlorid and 4 mg. of neoarsphenamin.

At the end of six months at least, the patient should rest entirely for from two to six weeks without treatment, in order to allow elimination of mercurial and arsenical products.

Effects of Intraspinal Treatment.—Besides the ordinary reaction familiar to us all, the writer speaks of:

1. The Subjective Order of Curative Effects. The symptoms disappear as follows: Lancinating pains, root pains, headaches, paresthesias, pareses, contractures and general mental symptoms.

2. Objective Order of Curative Effects: There is improvement in everything that does not remain as a result of an irreparable sclerosis, such as, impaired pupillary reactions, optic atrophy and absent knee jerks. The more recent and more active the process, the more likely the recovery. The author has seen a reappearance of knee jerks and pupillary reflexes. In general paretics, the Wassermann reaction on the fluid is the last to disappear; the Lange curve changes less quickly than the Nonne and Noguchi reactions. When the curve does change, it first becomes similar to that of cerebrospinal syphilis and finally it becomes normal.

Laboratory changes are always slower than the clinical, the latter frequently clearing up in from three to four months. The laboratory changes, therefore, serve as a guide for further treatment even after clinical symptoms disappear; patients with only slightly advanced cases of paresis usually require a year and a half of treatment, although they may show complete remission in nine or ten months; tabetics usually require from seven to eight months; advanced paretics of more than a year's standing may show temporary improvement but progressively get worse in spite of intensive treatment.

Because certain critics maintain that the alleged cures are nothing more than a coincidence of spontaneous remission accompanying treatment, the author feels obliged to discuss the question of so-called spontaneous remission.

Many paretics show spontaneous remission usually following variable periods of pseudoneurasthenia, followed by a period of excitement with delusions of grandeur or persecution. If treatment is resorted to before the spontaneous remission, it is easy to argue that one is not safe in ascribing the remission to the treatment, but the fact remains that many paretics do not show spontaneous remission. The writer quotes the percentages of Cotton and of Southard and Salomon which show certain advantages of treatment in spite of the fact that they reported nonselected cases. He is willing to admit that cases of more than a year's duration do not give a good prognosis, although the patients may be helped; but he does insist on the fact that patients found early, show complete remission of both clinical and laboratory symptoms, and this is the distinguishing feature between spontaneous remission and that due to treatment. In spontaneous remissions, the reactions on the blood and the spinal fluid usually remain positive, and if they show any change, this is very slight.

Real and Apparent Aggravation of Symptoms in Treated Cases.—General paretics ordinarily show a certain amount of mental confusion and increased difficulties in speech and in writing; tabetics show difficulty in walking and ataxia, but this usually disappears in from eight to ten days following injection. There are a few cases, however, in which aggravation of earlier symptoms is persistent. Sometimes certain infectious diseases, such as influenza, have a similar effect. The author had one tabetic whose symptoms were aggravated transiently just after vaccination.

Permanency of Results.—Nothing can yet be affirmed as intraspinal and intravenous methods have been in use only a few years. The author cites the case of a patient with paresis, first seen in December, 1916. All clinical and laboratory signs had disappeared by November, 1917, and had not reappeared when the patient was last seen in February, 1919, without any other treatment than an occasional intravenous and intraspinal treatment every three or four months.

The author then submits the history of treatment and results in the case of five general paretics, noting results as follows: One recovery; one improved but with very slow and mild progression; one almost recovered with great amelioration; one with unusual rapid clinical improvement; and one with rapid clinical improvement and modification of laboratory signs.

The conclusions which he reaches are based on the study of fifteen cases of general paralysis, eight of tabes, four of syphilitic spinalparaplegics, and two of syphilitic cerebrospinal syphilis. He omits his advanced cases of general paralysis and such other patients as were under his care for only a short period.

Conclusions.—1. The indication for exact intraspinal treatment is furnished by positive Wassermann reaction on the fluid and low Lange's colloidal gold reaction.

2. The efficacy of such treatment is strikingly manifested in those syphilitic processes which have not as yet involved the deeper portions (parenchyma) of the nervous system.

3. In general paralysis, such treatments do not give results if cases are of more than six or eight months' standing.

4. In tabes, results are almost always good; subjective symptoms disappear rapidly and a large number of the objective, likewise.

5. In syphilitic optic atrophy, intraspinal treatment is most favorable for arresting a rapidly progressive blindness.

6. In cases with radicular involvement, pains disappear much more quickly than under intravenous treatment.

7. In combined system diseases, especially sclerotics, treatment is of little value unless the cases are caught early.

8. Remissions which occur in cases of general paralysis may persist for years.

9. It is too early to maintain, because of the few years in which intraspinal treatment has been employed, that we may bring about definite cures in general paralysis although we may affirm that this may be the case.

10. Remissions due to intraspinal treatment differentiate themselves from spontaneous remissions in that the latter show only temporary amelioration of clinical symptoms, and laboratory symptoms still persist. In contrast to these, remissions due to therapeutic measures show true cures with amelioration of both clinical and laboratory symptoms but the touchstone of treatment in general paralysis lies in diagnostic skill which one may fortify by the aid of the five laboratory reactions.

12. The author's method, if followed with rigorous precautions, is harmless. These precautions include careful diagnosis of the case, and mild therapeutic measures, intravenous and intramuscular, to determine the patient's resistance to antisyphilitic remedies.

Jones, Detroit.

STUDIES ON EPINEPHRIN. I. EFFECTS OF THE INJECTION OF EPINEPHRIN IN SOLDIERS WITH "IRRITABLE HEART." J. T. Wearn and C. C. Sturgis, Arch. Int. Med. **24**:247 (September) 1919.

The authors report their results with the Goetsch test in cases of "irritable heart" of soldiers. Goetsch has reported that cases of hyperthyroidism give a very marked reaction to small doses of this drug. While a large number of these cases of "irritable heart" gave a positive reaction to the Goetsch test, studies on the basal metabolism and a careful clinical analysis of the cases do not lend support to the theory of hyperthyroidism as a causal factor in "irritable heart." The technic employed by Wearn and Sturgis in carrying out this test was as follows: The patients were put to bed at absolute rest for one hour, and at the end of this period control readings of the blood pressure, pulse and respiratory rates were made at five minute intervals. At this time note was also made as to the presence of nervousness, precordial pain, palpitation, dizziness or any other symptoms; likewise, the objective condition of the patient was observed, and the presence of any of the following signs was recorded: tremor of the hands, sweating, coldness of the hands, throbbing of the neck, epigastrium or the precordium, and pallor or flushing. At the end of the hour the patient was usually quiet, and the blood pressure and heart rate were normal (in contrast to the patient with exophthalmic goiter in whom a marked tachycardia persists even when at rest in bed). After satisfactory control readings were obtained, 0.5 c.c. of a 1:1,000 solution of epinephrin freshly prepared was injected deep into the deltoid muscle. The onset of the reaction occurs about twelve minutes after the injection, the height is reached on an average in thirty-two minutes, and the reaction finished in approximately one hour. A positive reaction is characterized by a rise in blood pressure of 10 mm. or more, associated with a rise in pulse rate of at least 10 to the minute (provided that these are not isolated rises), and is associated with tremor, pulsation, nervousness, precordial pain, dizziness and restlessness. Cases with a fairly marked rise in blood pressure and pulse rate without other changes were also regarded as giving positive reactions.

Twenty-six normal soldiers were used as a control, and in not a single case was there a positive reaction to epinephrin, as judged by the rule formulated to determine a positive or negative case. The blood pressure and pulse curves showed slight, if any, change, and the characteristic subjective and objective conditions of the epinephrin sensitive group were lacking. In distinct contrast to the epinephrin sensitive patients, with marked restlessness and anxiety, the controls remained perfectly quiet, some of the men reading throughout the greater part of the test, while others actually slept.

Of seventy-three cases of the group of soldiers with "irritable heart" whose symptoms were of long standing, Wearn and Sturgis found that forty-three, or 59 per cent., showed a definite hypersensitiveness to epinephrin or gave a positive reaction as described above. Twenty-seven, or 37 per cent., showed no reaction. Three cases, or 4 per cent., gave a doubtful reaction. Of eighteen cases of "irritable heart" in which the symptoms dated to some recent infection, such as rheumatic fever or pneumonia, six gave positive reactions to epinephrin and twelve gave no reaction. Observations made on a group of cases in which the symptoms developed following some nervous or physical strain, showed, that of seven of this type, five gave a positive reaction and two gave no reaction. The proportion of positive reactions in cases of "irritable heart" is in distinct contrast to the complete absence of positive reactions among normal, trained soldiers.

The authors suggest that the symptoms and signs induced in the positive cases are the result of a hypersensitive, sympathetic, nervous system. This belief is based on the fact that epinephrin has a selective action on the sympathetic nervous system. They state that a careful clinical analysis of the patients showing positive and negative reactions revealed no essential clinical differences between the two groups. They are unable to state that the clinical condition of the patients who are sensitive to epinephrin depends in any way directly on the factors underlying the positive reaction, or that the factors causing the symptoms in the positive and negative groups are the same. Although all efforts were made to improve the physical condition of these men, not one of those who gave a positive reaction, and only three who gave a negative reaction, were able to do full duty. The authors conclude that this class of patients is not a true product of the war, and that they are seen not only in military but also in civilian life. They are the type that go from one outpatient clinic to another with complaints of nervousness, weakness, palpitation, precordial pain and dyspnea; they are classed as neurotic or neurasthenic individuals and have usually been receiving medical attention for years. There seems to be a certain level of strain below which these people can live without symptoms, and their one effort in life is to keep below this level, above it their symptoms appear and they break.

STUDIES ON EPINEPHRIN. II. THE EFFECTS OF EPINEPHRIN ON THE BASAL METABOLISM IN SOLDIERS WITH "IRRITABLE HEART," IN HYPERTHYROIDISM AND IN NORMAL MEN.

E. H. Tompkins, C. C. Sturgis and J. T. Wearn, Arch. Int. Med. **24**:269 (September) 1919.

Four groups of subjects were studied. Two of these groups were negative to epinephrin and showed no reaction, while two were positive to epinephrin and responded with the typical reaction of the hypersensitive subject. The groups positive to epinephrin consisted of patients with "effort syndrome" ("irritable heart") who reacted to epinephrin and a small series of cases of hyperthyroidism. The groups negative to epinephrin consisted of normal subjects and patients with "effort syndrome" who did not react to epinephrin.

With unbroken regularity the gaseous metabolism showed a rise after the injection of epinephrin (0.5 c.c. of 1:1,000 epinephrin solution). This is true in the normal, as well as in the pathologic cases, and whether there is any other sign of reaction or not. This increase in metabolism, however, is far more marked in cases with a positive than in those with a negative reaction, and it is parallel to the pulse, blood pressure and symptomatic changes. With one exception, every positive reaction was accompanied by a rise in metabolism of 20 per cent. or over. On the other hand, no negative reaction showed a rise above 20 per cent. and in the majority of cases not above 15 per cent.

The increase in the metabolism is expressed partly by a greater oxygen deficit in the expired air, but far more by an increased amount of air breathed, as is shown by the greatly augmented minute volumes. The increase in the minute volume is as invariable as is the rise in metabolism. The amount of air breathed per minute was increased as much as two liters in a considerable number of cases. There is an increase of the respiratory rate or depth, or both.

There is frequently an elevation of the respiratory quotient. The maximum of this elevation normally precedes that of the metabolic increase. The increase in metabolism is not due to excitement attendant on injection or the stimulation of heart and respiratory muscles resulting from injection. (The basal metabolism in cases of "irritable heart" with increased respiratory activity shows no changes, and after injection of atropin, although the pulse rate is increased, no metabolic increase occurs.)

The authors conclude that epinephrin acts indirectly in causing an increase in the metabolism, and it is by acting through the sympathetic system that it produces the changes—increased muscular tonus, tremor and hyperglycemia—which are probably the actual causes of the increased metabolism.

Finally they conclude that as epinephrin is a stimulant to the sympathetic system, the differences in reaction and metabolic response in the negative and positive cases are probably due to differences in that system.

MYERS, New York.

CONTRIBUTION TO THE STUDY OF AUTOMATISM IN THE MEDULLA. ABRAMO LITVAK, Riv. di patol. nerv. **24**:15 (March 25) 1919.

The author refers to Luciani's law that the excitement of centripetal nerves may awaken flashes of consciousness and motor impulses, preceded by the choice of centrifugal pathways through which the stimulus is transmitted, and also to Wundt's conception that tendon and superficial reflexes are voluntary movements become automatic by filogenetic development. The finding of nerve terminals in tendons disposed of Westphals conception that tendon reflexes were due to direct stimuli applied to muscles through their tendons. With these preliminary remarks, the author introduces a new abnormal plantar reflex. His discovery disproves the contention of Marinesco that in paralysis of the lower extremities the extensor center alone remains functionating because in his cases (Figs. 2 and 3) both the extensor and flexor abnormal plantar reflex were demonstrated. While he has noted the abnormal flexion of the toes caused by stimuli applied to the tendo achilles, thigh, abdomen, etc., in cases of hemiplegia due to capsular or cortical lesions, his sign has been observed only in lesions of the peduncles above the fibers for the arm. This is an important localizing sign. However, the toes occasionally assumed the extensor Babinski type and the reflex was not always in flexion. In many normal persons and in hemiplegics with lesions in other sites, grasping the paretic hand never caused either the author's sign or the Babinski phenomenon.

Case 1 illustrates a vascular accident to the left side of the pons; therefore all the abnormal signs must originate in the medulla. The "sincinesie" and associated movements in these cases depend on the intensity of the stimulus, the condition of the centers receiving it, the length of the pathway traversed and the resistance offered by it. Babinski long ago noted that "sincinetic" movements were more readily brought out by stimulating the paretic limb. The author noted a diffusion of these associated movements from the big toe to the feet, leg and thigh, and while these movements always spread from the upper to the lower (from bulb to cord) extremity, the contrary is never true. This bears out Sherrington's law of caudal radiation of spinal reflexes in mammals. In the author's case, it was always possible to provoke movements in the lower extremity by strongly applied or painful stimuli to the upper extremities, but never if the stimuli were applied to the lower extremities. In this case both the flexor (author's sign) and extensor responses of the toes were obtained. The flexion of the toes of the paretic foot was obtained either by (1) reflex stimuli applied to the other thigh or (2) strong pressure applied to either the sound or paretic hand. The extension of the toes of the paretic foot (right) was obtained by stroking the plantar surface (Babinski sign) of the paretic foot, or by applying the stimulus to the thigh of the paretic leg (Oppenheim).

The author's patient also had a paralysis of the left sixth cranial nerve and a complete hemianesthesia to all types of sensibility; also a bilateral clonus and absent left plantar reflex. All abdominals were absent.

By applying a mild stimulus, extension of the toes (Oppenheim) was obtained; by pricking more strongly the paretic thigh, flexion of the toes was obtained, even if this was done to the arm or abdomen of the same side. When the left (sound) leg was pricked, the right paretic leg was always withdrawn. The author believes that the flexion reflex of the toes of the paretic side was pathologic and like all defense reflexes had its center in the medulla because: 1. There was always greater response in flexion or in extension when the plantar surface of the paretic leg was stroked corresponding directly to the strength of stimulus than was obtained by the same stimulus in the other leg. 2. There was no paralysis of the left leg. 3. All the abnormal associated movements were very active on the right and hardly elicitable on the left side. 4. Flexion of the toes on the right was obtained by stimulating other regions of the right extremities. This flexion reflex in the toes, like the retraction in flexion of the paretic leg, is a medullary reflex function. Because this reflex is objectively like the normal, it might be difficult to determine in a given case by this alone which is the paretic side.

In the author's second case, attention is drawn to Dagini's two signs; the oculocardiac reflex and the adduction of the paretic arm on pressure of the eyeball.

The author's third case illustrates his point that the abnormal associated movements seen in paretic limbs are present in direct proportion to the degree of paralysis, that is, the condition of the center receiving the stimulus. For that reason neither the abnormal extensor Babinski or flexor response was obtainable in the right paretic leg because this was little involved. The normal plantar reflex was absent, but when the eyeball was pressed, the pulse slowed up and the paretic arm was adducted showing that the automatic action of the medulla was free from cerebral inhibition through the corticobulbar and corticospinal tracts.

The next part of the author's extensive article is devoted to discussion as to what influence mild pyramidal lesions, as shown by the Babinski sign, and severe lesions, as shown by the Marie Foix sign, have on the automatic functions of the medulla.

The Babinski phenomenon is elicited not only by plantar stroking but also by stimulating various antagonistic groups of muscles of the paretic leg and even of the sound leg. Babinski has always contended that this phenomenon could coexist with a normal plantar flexion of the toes. Eight cases are briefly cited to show the variations in plantar reflexes in various types of hemiplegia and paraplegia, varying from complete absence to the Babinski phenomenon with flexion of leg and thigh. When the normal plantar reflex mechanism is absent independent centers in the cord react to cutaneous, osseous, joint and tendon stimuli, and responses are obtained from the toes even when the stimulus is removed to the leg, thigh and even the upper extremity. The classical, most frequent response is extension of the big toe, but at other times there is abduction of the toes or fanning. According to the author, the Babinski reflex, therefore, is not a plantar reflex but an expression of an abnormal reaction to stimuli, extending over a large zone. If this is true, then the flexion of the big toe provoked by stroking the thigh and the abdomen and by the author's method of squeezing the paralyzed hand, stretching the achillis tendon, etc., are not at all plantar reflexes but so-called defense reactions, improperly so designated, which have their center in the cord, bulb or subcortex, and not in the cortex which is the site of the mechanism of the normal plantar reflex.

The author believes, with Goldflam, that the center for the Babinski reflex is in the lumbosacral cord, and does not operate until the corticospinal inhibitions are removed. It has been noted in complete transverse lesions of the cord above the lumbosacral level, never when this is crushed. The fact, that the Babinski reflex is the normal response in the first year of life, is proof that it is a spinal function. In infants the reflex zone for this reflex is also wide, as it is also for the abdominals and the cremasterics. The author's twelfth case strengthens his belief that the Babinski is a spinal reflex. This patient had a tetraplegia with complete loss of all types, of sensibility and bilateral Babinski reflex. At necropsy, fracture of the second and third cervical vertebra with complete severance of the cervical cord was found.

Retraction of the leg (Marie Foix sign) is caused only by strong painful stimuli which contract the sartorius, adductors of the thigh and the anterior tibial group, or occurs in very profoundly paralyzed limbs. The author agrees with Marinesco that the Babinski phenomenon is the first to appear and the last to disappear. When the patient is dying or his medulla has become almost completely removed from the cerebral control by profound paralysis, stimuli produce a more widespread reaction of medullary and cord centers resulting not only in extension of the big toe, but also in retraction of the limb.

The last chapter deals with the nature of reflexes seen in moribund hemiplegics and paraplegics.

The sign of Marie Foix (described in 1853 by Sherrington in the spinal frog) is not so commonly seen as the Babinski, and is most commonly seen in the profoundly paralyzed. It occurs usually as the Babinski reflex disappears, and persists until death. Occasionally the Babinski reflex is seen in the nonparetic limb and the Marie Foix sign on the paralyzed side; or a mild stimulus applied to the paretic leg produces a Babinski reflex, a stronger one causing the latter. Observations in many cases convince him that the Babinski reflex is a sign of milder pyramidal tract disturbance than the Marie Foix sign. In the presence of cortical and meningeal lesions other than the pyramidal tract injury this sign, however, loses its prognostic value. As an illustration, the author quotes a case of delirium with right hemiparesis, Babinski reflex on the right, Marie Foix sign on the nonparetic, left side. When delirium disappeared, the Marie Foix sign disappeared. The case was one of status epilepticus attacks with delirium at intervals of a few months, following a frontal skull injury which left him with a right hemiparesis. Another sign of profound paralysis, usually with a fatal ending, is the Dagnini "trigeminal-brachial" sign of adduction of the paretic forearm alone or with flexion of the forearm and hand on the arm. Dagnini's oculocardiac reflex is frequent in the young, but never seen in the aged. In many paralyzed patients the independence of the medulla from cerebral control favors the exhibition of retardation of this reflex. The author's Cases 13 to 19 illustrate these points. They are accompanied by graphic tracings showing the retardation of the pulse in the application of the oculocardiac reflex. In many of the patients showing the Dagnini and Marie Foix signs, there was also Cheyne-Stokes breathing which he also classes as an "ultimum moriens" reflex. The author in his discussion of these cases mentions another sign of Dagnini's, that is, the "frontalis" sign. The author declares that in supra bulbar pyramidal lesions the upper branch of the facial nerve also is involved with smoothing of the forehead on the paralyzed side (Case 19).

Osnato, New York.

ETUDE SUR L'ORIGINE ET LA NATURE DE LA SCLEROSE EN PLAQUES. G. Marinesco, Rev. neurol. **26**:481 (June) 1919.

There are two leading conceptions of the etiology and the pathology of multiple sclerosis: First, that it is inflammatory in origin, or vascular; second, that the inflammatory process is due to an infectious agent. It is not so much a sclerosis of nerve tissue or centers as it is a disseminated inflammation. It is especially in the acute and the subacute forms that the inflammatory character of the disease is best seen. The theory that the vascular lesions are secondary to those in the nervous tissue can no longer be accepted; on the contrary, the reverse is true, in spite of the earlier teachings of Charcot, Bourneville, Wernicke and others.

It is the opinion of Pierre Marie (and this is now accepted by Wernicke, Marburg and others) that the vascular lesion is in the form of a disseminated arteritis, the sequela of some well advanced primary condition, such as scarlet fever, pneumonia, erysipelas and similar diseases. Sometimes the arteritis appears toward the close of the conditions, or during convalescence, or even when recovery seems to be absolutely complete.

The lesions localize themselves by preference in the white substance, and follow the course of the smaller vessels, especially the veins. This explains the relative integrity of the nerve cells, but even these may be seriously damaged in a rapidly advancing case. The essential point to remember is that there occurs an infiltration, in and about the adventitia and the perivascular tissues, of lymphocytes, plasma cells, polyblasts and occasionally mast cells. Polymorphonuclears are the exception, and eosinophils do not occur. The proportions of these infiltrative elements vary with the stage of the lesion. In the chronic or slowly advancing processes, lymphocytes and plasma cells predominate and should not be confused with the inflammatory type showing macrophages in the vessel walls. The chronic type shows changes not unlike those in general paresis; the inflammatory type follows only the disintegration of myelin into fatty bodies. Simultaneous with the infiltration of the vessel walls is the growth of the neurogliar cells. Giant neurogliar cells are found particularly in the cerebral cortex and in the optic nerve; and possess a marked capacity for fibrillogenesis.

The perivascular infiltrations are frequently nodular and not unlike lesions in syphilis, especially in general paresis. The adventitia does not constitute a barrier to the inflammatory process, as is evident from the invasion of lymphocytes, plasma cells, and polyblasts into the surrounding tissue.

The writer had two cases which showed inflammatory processes in the following important regions: periventricular, periependymal, and about the aqueduct of sylvius. More rare is an involvement at the level of the fourth ventricle or in the ependyma of the cord. This statement is in agreement with the opinion of other writers cited, and might lead to the conclusion that the spinal fluid is the agent through which infection reaches the areas just mentioned, and that the virus gets to the fluid through the medium of the lymph paths. Consequently, wherever the vessels and their corresponding lymphatics are involved, there will be a lesion of nervous tissue. This explains the frequent involvement of the optic nerve in multiple sclerosis.

Marinesco's studies do not lead him to accept Ribbert's conclusion that vascular thrombi occur as the result of inflammatory processes in multiple sclerosis. The intima of the vessels, like the gray matter of the brain and cord, appears to escape. The virus in the lymphatic circulation affects chiefly the nutrition of the myelin, but also may bring about a secondary degeneration of the axis cylinder, although even in this case there is usually collateral regeneration.

The remissions and exacerbations of multiple sclerosis are more in harmony with the theory of an infectious process than a toxic (due to lead, mercury or other minerals). While fever and chills in certain cases have led certain authors to look on multiple sclerosis as due to influenza and other similar diseases, it is more likely that it is a specific infectious disorder, and while similar in certain respects to syphilis (particularly general paralysis), yet it is evidently not due to the spirochete of syphilis. Although Nonne in a study of seventeen cases of multiple sclerosis obtained a positive Wassermann reaction in three cases, the fact remains that neither clinical nor histologic examination points to syphilis in multiple sclerosis. Moreover, certain writers, like Collins, who give a routine Wassermann test in all their cases of multiple sclerosis, report all reactions negative.

It was not until September, 1917, that Kuhn and Steiner, by injecting the blood and spinal fluid of patients affected with multiple sclerosis into guinea-pigs and rabbits, demonstrated a spirochete in rabbits. They injected blood, spinal fluid and a mixture of both into the peritoneal cavity, the heart and the cavity of the eyeball of these animals, and obtained, in from three days to two weeks, disturbances of motility, especially a staggering gait, more pronounced in the hind limbs. This condition lasted from a few hours to nine days, and in the fatal cases was followed by paralysis and death. Examination of the blood drawn from the eye or ear of the animal revealed the presence of spirochetes which, in form and size, resembled those that produce hemorrhagic icterus. They were spiral in form and often showed a refracting point at the extremity. They were also variable in form, and either extremity might be provided with a hair-like prolongation. In the multiple sclerosis of man, they have not seen the spirochete.

By subdural injections, Simon has demonstrated the transmissibility of the virus in rabbits and has observed paralytic manifestations followed by death. In March, 1918, Siemerling announced that by the aid of the ultramicroscope, he had found spirochetes in the brain of a patient that had had multiple sclerosis. They presented the same characteristics as those described by Kuhn and Steiner.

In October, 1918, Marinesco began experimental work with two patients who showed many of the clinical symptoms of classic multiple sclerosis. He made intracerebral, intraperitoneal and intraspinal injections of the spinal fluid of these patients into six guinea-pigs. Two of the pigs showed, three or four days after the injection, motor disturbances in the posterior portion of the body, especially in the form of disturbed gait. Spinal fluid was then withdrawn from the fourth ventricle of these animals and a considerable number of spirochetes, like those described by Kuhn and Steiner, were seen under the microscope. M. Pettit and Marinesco himself, in certain later attempts at transmitting the virus, obtained negative results. This proves only that the parasite is not always in the spinal fluid, or if it is there, it is present only in reduced numbers. Moreover, the two patients from whom the inoculating fluid was drawn were in a more severe and advanced stage of the disease; as a consequence, the parasites were probably less numerous and virile.

Summary.—While the research is still incomplete, it brings to light two new facts: 1. Multiple sclerosis is transmissible due to the virus of a living organism. 2. This virus is the product of a spirochete entirely distinct from the *Spirochaeta pallida.* These facts on first consideration do not seem to harmonize with the isolated character of the disease which is neither endemic or epidemic, nor is it contagious; and it appears to confine itself to the nervous system.

Reviewing all the facts, however, multiple sclerosis appears much more like an exogenous malady of infectious nature, than like an endogenous malady as maintained by Strumpell. The course of the disease, the successive exacerbations, and the appearance of the fundus permit us to differentiate, in the living subject, the various phases of the disease (really stages of development of the parasite) which are not unlike those of syphilis, and lead us to conclude that the spirochete observed by Kuhn and Steiner, and by Marinesco is the pathologic agent of the disease. Multiple sclerosis, then, presents a series of well defined symptoms, and though varying in their clinical aspects, is a true nosologic entity and not a syndrome of sundry causes.

Jones, Detroit.

SCHIZOPHRENIA AND CHOROID PLEXUS. C. von Monakow and S. Kitabayashi, Schweiz. Arch. f. Neurol. u. Psychiat. 4:363, 1919.

The writers call our attention to the fact that, until recently, we have attributed the normal and abnormal manifestations of the mind to a function of the cortex alone, and have looked on the secretory organs much as we have on the circulatory apparatus, that is, as a secondary source of assistance. It would be more logical to attribute our psychic life, consciousness and affect to a more closely associated function of these two structures. This view has phylogenetic and autogenetic support. In studying serial sections of a four weeks old human embryo, we find that those portions of the dorsal alar laminae which subsequently become differentiated to form the choroid plexus, particularly in the vicinity of the vagus and glossopharyngeal centers, is always well developed; in like manner we find this structure developing in the median portion of the hemispheres. All of this proceeds with great rapidity, long before the essentially nervous elements of the medullary structure reach a corresponding degree of differentiation.

The writers look on this as a sort of overture for the later physiologic interchange between the choroid plexus on the one hand, and the rest of the brain on the other. In other words, the embryonic relation itself suggests the important rôle of the choroid plexus. The other glands of internal secretion also, particularly the suprarenals have, at the corresponding period of evolution, reached a surprising degree of differentiation. In short, the endocrine system is well developed at a period of fetal life when the essential structures of the brain are still lying in deep slumber. We can look on the organs of internal secretion as the mother source of our affects and instincts. In the grown up person, the choroid plexus in the fourth ventricle, as well as in the lateral ventricles, appears to be correspondingly less well developed than in the fetus; in lieu of this, however, they are much more intricately folded and villose. It has been demonstrated that the choroid plexus is a true gland, which not only secretes cerebrospinal fluid, but also neutralizes toxins derived from other glands and circulated in the blood. According to the writers, the substances so noxious to the brain are particularly epinephrin, thyroglobulin, and the secretions from the hypophysis, the epiphysis and the gonads.

The morphologic basis of the psychoses, particularly schizophrenia, has in times past been looked for and partly found in the cortex, while the internal secretory glands, particularly the choroid plexus, have been disregarded. The sources of our affect-life must be sought for primarily, not so much in the field of brain mechanics, as in that of biochemistry; those tissues of the brain must be subjected to the greatest morphologic changes which are most intimately related to the finer processes of nutrition and to the chemical changes that afford protection against substances circulating in the blood. These consist of the tela choroidea, plexus choroideus, the ependyma and the protective glia covering. The writers accordingly look on the choroid plexus, in the typical forms of schizophrenia, as not only functionally but also morphologically drawn into the circle of pathology.

Up to date, seventeen pathologic brains as well as a larger number of normal brains, have been studied. The former comprised not only cases of schizophrenia, but also other types of mental disturbances, such as progressive paralysis, tumors, etc. The authors were greatly surprised to find that the choroid plexuses of twelve patients, who had for years suffered from the more severe schizophrenic symptoms, were not found normal in a single instance. The results can be summarized somewhat as follows: hyperemia, diffuse degeneration of the villi, amyloid degeneration of the connective tissue, accumulations of colloid masses within the plexus and interpapillary exudation.

In other psychoses, not schizophrenic clinically, somewhat similar changes were found, but these were of much less intensity. Hyaline degeneration and a moderate atrophy of the choroid villi were observed in a few senile individuals and in chronic alcoholics. In a number of cases of schizophrenia, there were marked hyperemia to the point of capillary blood extravasations, thrombosis, and increase in the peri-capillary connective tissue with hyaline degeneration to the extent of complete obliteration of the villi. The pathologic process usually seems to begin with a cell infiltration in the tissue between the capillary walls and the secretory cells.

In view of the changes noted in the brains of schizophrenic patients investigated up to date, there remains little possibility, in contradistinction to other psychoses, that recovery without permanent defect may take place. The most serious consequences of a primary or even a secondary disease of the choroid plexus, the writers view in relation to the physiologic processes, since the innate protective power and resistance of the choroid plexus to substances harmful to the brain is lowered, and probably permits greater filtration, resulting in chemical combinations with the brain tissue itself, through the medium of the cerebrospinal fluid, against which the protective membrane of glia tissue is inadequate. Through disease of the plexus by way of intoxication, a disease of the cortex is eventually added; this would open a way for the development of typical schizophrenic phenomena.

In conclusion, the writers state their belief that this view would bear out the principle of minute division of labor, with the nervous system on the one side, and the glandular system on the other; and that a united action of the biophysic functions of the cortex and the biochemic functions of the glandular system would result in creating the highest phenomena of our psychic life.

Woltman, Rochester, Minn.

MANIFESTATIONS NERVEUSES DU TYPHUS EXANTHEMATIQUE: 1. Reactions Meningees Tardives, II. Inegalite Pupillaire, III. Nevrites et Polynevrites. Demetre E. Paulian, Rev. neurol. **26**:650 (Aug.) 1919.

The author discusses the subject under these heads: I. Late Meningeal Reaction of Exanthematous Typhus.

In an earlier study the author showed, in collaboration with Devaux and Tupa, that there is always a marked meningeal reaction in the first days of the infection. He has also been able to establish an almost constant cytologic formula for this disease at its crisis and during the period of convalescence.

During the first few days of the fever, the spinal fluid on puncture shows an increase of pressure, and drainage has a salutary effect on the patient.

In a few grave cases with marked agitation and prolonged confusion, the spinal fluid is xanthochromatic.

All the fluids examined show a large number of cells, but no bacteria or parasites.

The cell changes are:

1. From the second to the fourth day there are lymphocytes, round cells, easily stained by basic dyes, the nucleus filling the entire cell body.

2. On the fifth day (when the patches appear) there are large mononuclears which persist during the period of eruption. The nuclei of these mononuclears may be eccentric, or only slightly so. There may be a few polymorphonuclears. They increase as the disease progresses.

3. About the twelfth day the mononuclears are replaced by lymphocytes, at the beginning easily stained and persisting well into convalescence, especially in cases in which the nervous system is involved; in some cases with nervous complications this condition may persist for even a year and a half.

Without the aid of the Wassermann test it would be difficult to distinguish some of these postexanthematous cases from syphilitic cases. In the typhus cases there is always a high albumin content (as much as 0.50 or 0.65). Gentle heating in a test tube will produce a slight opalescence or milky whiteness.

A late lymphocytosis in the fluid is an important diagnostic feature and almost pathognomonic in individuals with nervous complications and especially in postexanthematous psychoses in which the spinal fluid (even in high quantitative tests) proves negative.

II. Cases in which There Are Inequality of the Pupils.

About 60 per cent. of patients show unequal pupils. If these patients are seen a considerable period after the exanthem has disappeared, one may mistake them for syphilitics. In the exanthematous cases the fluid, however, is always negative. The cytologic phenomena just described help clear the diagnosis, and necropsies on typhus patients reveal lesions of a diffuse leptomeningitis not unlike those of a rapidly coursing case of general paralysis.

The constant presence of a lymphocytosis is proof of the meningitic involvement.

III. Postexanthematous Neuritides and Polyneuritides.

Motor, trophic and sensory disturbances of the peripheral nerves may occur during, or after, the course of typhus. The writer has noticed such disturbances in fifty-five cases. The position in bed, trauma sustained during excitement of the delirium, and similar factors may determine the site and nature of the neuritis.

Subjective as well as objective symptoms may be prominent. Pains in the legs may produce functional disturbances in gait. The circumflex nerve is frequently involved, often owing to pressure of the shoulder on the bed. Mechanical restraint during delirium frequently results in an ulnar neuritis. Various inclemencies, including cold, heat and humidity, may be regarded as adjuvant causes.

Spasticity may result from pain and consequent disuse. The writer has also observed paresthesias, objective cutaneous disturbances of sensibility and modifications of electric reactions.

Bacaloglu has described a case of paralysis of the soft palate identical with diphtheritic paralysis.

The writer enumerates a variety of cases which have presented themselves at his clinic; one showed an atrophy of the infrascapular muscles; another a bilateral facial neuritis (which he calls diplegie faciale nevritique). He has observed three such cases, one not showing itself until nine months after the exanthem.

The etiologic factor is evidently toxic. But the toxins elaborated, though not yet known, are evidently carried by the vessels, and thus arise the vascular lesions such as phlebitides, and arteritides, which easily explain the myalgias and subsequent gangrenes. From the vessels the lesions extend to the peripheral nervous tissues, and we thus have the various neuritides and polyneuritides already described.

Jones, Detroit.

ON THE GENESIS AND PHYSIOLOGICAL SIGNIFICANCE OF SPASTICITY AND OTHER DISORDERS OF MOTOR INNERVATION: WITH A CONSIDERATION OF THE FUNCTIONAL RELATIONSHIPS OF THE PYRAMIDAL SYSTEM. F. M. R. Walshe, M.D., Temporary Major, R.A.M.C., Brain **42**:1 (April) 1919.

In a previous paper, Walshe concluded from a study of certain of the reflex phenomena seen in the lower limbs in spastic paralysis, that "analysis of the phenomena of spastic paralysis of the lower limbs in man demonstrates the close analogy existing between the physiological principles of motor innervation in animals and in man."

The author resumes the subject and discusses spasticity and decerebrate rigidity, Förster's theory of the origin of spasticity, the proprioceptive system of the cerebellum, associated movements in hemiplegia, three components in motor innervation, tremor-rigidity syndrome of paralysis agitans, other organic cerebral lesions, and cortical lesions and spasticity.

In the author's previous paper evidence was advanced that in spastic paralysis of the lower limbs there were two clinical types, the extended and flexed; and that in hemiplegia or in any spastic paralysis due to a lesion confined to the pyramidal system, the extended type was constant irrespective of the degree of pyramidal involvement. Walshe believes that both anatomic and physiologic considerations indicate that decerebrate rigidity is dependent on the removal of a crossed cerebral control.

As to the special position of the upper limbs in man, the view advanced in the present paper is summarized by the author as follows: "The spasticity of spastic paralysis as seen in hemiplegia and in paraplegia in extension is analogous with the decerebrate rigidity of Sherrington. It is, therefore, an expression of the activity of the proprioceptive system taken as a whole. This system induces a tonic reflex, which, unmasked in the decerebrate animal, has been shown to possess a significance of a postural reflex—reflex standing. In man also we see in hemiplegia a rigidity which has the same significance. However, in this instance, the upper limb shows typically an attitude of flexion. The hypertonus is resident in the limb flexors, not in the extensors as in the lower limbs. This may be traceable to the altered function the upper limb exercises in man, and to its complete lack of the locomotion function."

Walshe criticizes Förster's theory of the origin of spasticity and states that in each element Förster's hypothesis is incompatible with the facts.

Relative to the proprioceptive system and the cerebellum, he summarizes his views as follows: "Viewed in the light of Jackson's teaching and the physiological researches of Sherrington, there appear good grounds for regarding the cerebellum as an important link in the reflex arc subserving the rigidity of hemiplegia. We may, then, look for the 'complimentary inverse' of spastic paralysis of cortico-spinal origin in lesions involving the cerebellum. Both clinical and experimental observations demonstrate how completely these lesions may produce such a contrasted picture. In that the cerebello-rubrospinal path appears to be the efferent limb of the proprioceptive reflex arc, we may regard the motor disorders following its inactivity, which cerebellar disease entails, as an example of extrapyramidal disease."

Walshe ventures to suggest an analogy between the associated movements of hemiplegia and experimental red nucleus reactions. He says "if, as in hemiplegia, one corticospinal path is out of action, then control over the crossed postural reflex is in abeyance, and on voluntary movements of the intact path of the musculature, especially forceful movements, such as demand widespread synergic fixation, there occurs the usual bilateral postural adaptation through the action of the intact proprioceptive reflex system. Freed from cerebral control on the paralyzed path of the body, this is manifested as a crude, associated movement which thus consists in a transient modification of the spasticity."

The author criticizes Holmes' and Wilson's views of the cause of tremor and rigidity, characteristic of paralysis agitans and certain basal and midbrain lesions. He says that "we may conclude that the view attributing the tremor and rigidity under discussion to uncontrolled cortical action is not established, and is contraindicated by such knowledge as we possess of the normal activity of the motor cortex."

Concerning the theory elaborated by Ramsay Hunt to account for the paralysis agitans syndrome, he states that the hypothesis is quite incompatible with what we now know of the physiology of such reflex acts as progression and muscle tone and, therefore, the theory of the functions of the corpus striatum, erected on the localization of these symptoms to a degeneration of this structure in a case of juvenile paralysis agitans, cannot be entertained.

Finally, he states that from clinical and experimental sources come several indications, as yet imperfectly understood, that a pure cortical lesion of the pyramidal system often does not produce spasticity as an accompaniment of the loss of power. They suggest the possibility of an interruption of another descending path than the corticospinal in the production of spasticity. Such a path need not necessarily be cerebrospinal.

Pollock, Chicago.

THE PATHOLOGICAL EXAMINATION OF FORTY INTRACRANIAL NEOPLASMS. J. Godwin Greenfield, M.D., M.R.C.P., B.Sc., Brain **42**: 29 (April) 1919.

Greenfield reports the examination of a number of intracranial neoplasms from the pathologic laboratory of the National Hospital for the Paralyzed and Epileptic. The study is purely pathologic and no attempt has been made to link together the clinical symptoms and the pathologic findings. The chief object of the report is to make an attempt to clear up some of the anomalies of nomenclature and to discuss in relation to the findings in the cases examined, the nature of certain groups of tumors peculiar to the nervous system. The paper does not lend itself well to abstracting, and should be read in the original form.

Of interest is the analysis of the relative proportion, age incidence and rate of growth of the various forms of tumor encountered in this series. Tuberculomas total 5 per cent., and sarcomas 5 per cent., while granulomas account for only 2.5 per cent. The author states that this percentage obviously has no relation to percentage of incidence, but is due to the low rate of mortality in the granulomas and tuberculomas, and to the fact that sarcomas are more usually encountered in general than in neurologic hospitals. The group of endotheliomas includes 25 per cent., and perivascular sarcomas 7.5 per cent. In all, mesoblastic, intracranial tumors form 40 per cent. of the series. Gliomas total eleven, of which three are multiple. There were three neuroblastomas and one ganglioneuroma. Thus, glia and nerve cell tumors form 37.5 per cent. Lastly, there were six cases of acoustic nerve tumors, comprising 15 per cent. of the series.

While generalizations from such a limited number of cases are unjustifiable, the author deems it worth while to note the age incidence and duration of symptoms of the various forms of tumor. Both cases of tuberculoma were in young subjects, and gave a fairly short history. The sarcomas occurred one at the age of 11, and one at the comparatively advanced age of 54. In these cases the history was a matter of a few months. All the cases of endothelioma occurred after the third decade, with the exception of the two cases of myxo-endothelioma which occurred in children of 11 and 13, respectively. In these patients the symptoms appeared three and four years before admission to the hospital. The other endotheliomas gave histories dating back from six months to six years. In the perivascular sarcomas, the author concludes that we are dealing with more malignant tumors but in one case the symptoms appeared probably five and certainly three years before admission. The gliomas of the series all gave a very short history, amounting in only one case to two years before admission and in the others to six months at most. The tumors of the acoustic nerve had histories of moderate length—the shortest, six months, was in a case of multiple tumors; the longest was two years.

No attempt will be made to summarize the views of the author on the nomenclature and classification of intracranial tumors because such a summary cannot be made and do justice to the author's opinions.

Pollock, Chicago.

THE PSYCHONEUROSES AND INTERNAL SECRETIONS. I. S. Wechsler, Neurol. Bull. **2**:199 (May) 1919.

The author expatiates on the as yet unsolved problem of the treatment of the psychoneuroses and points out how little we really know about it, despite the fact that various attempts have been made to cope with the subject. He finds that most efforts are directed at the problem after it has become one, and he points to the internal secretory disorders as a possible method of attack before its incipiency. In the attempt to give a possible physical basis to the psychoneuroses and looking for a "little more solid ground to stand on" he turns to the internal secretions for an explanation of the symptoms in some cases. He does not lay claim to any originality but merely tries to correlate a few well-known facts with other facts equally well known. He calls attention to the danger of overestimating the importance of the "facts" of internal secretions, since we as yet know so little about them. Nevertheless, he thinks that we have before us a fruitful field for investigation.

After reviewing briefly the knowledge that has thus far been accumulated, he tries to correlate in a general way the symptoms of early disturbance of the glands of internal secretions and the common symptoms of some of the psychoneuroses which bear very close resemblance. Then he quotes two typical cases of psychoneurosis, another of anxiety neurosis, and one of hysteria, in all of which he found marked signs of disturbances of the pituitary and in which treatment along that line gave favorable results. One obsessional case and two of marked anxiety he quotes as having shown distinct thyroid symptoms, treatment for which cleared up the neuroses. One case is correlated with the suprarenals because the history of the neurosis bears close resemblance to that of an "early Addison," another one is linked with the sex glands and, finally, one case is put into the class of general vegetative nervous system disturbance—the vagotonia of Eppinger and Hess.

Although the author is aware of "the great importance of environmental influences of social maladjustments, of psychic and emotional conflicts," he seeks in "disturbance of the glands of internal secretions a source of rational therapy" for some of the cases of psychoneurosis and tries to put them definitely in a group which might well be amenable to treatment. One cannot, of course, quarrel with the author for his attempt, and he is careful to point out that success in treatment cannot be cited as proof of cure, inasmuch as it is difficult to dissociate the influence of the personality from the "effect" of a drug, but it is well to emphasize the meagerness of the facts at our command and the great danger of speculation which the fascinating study of the internal secretions invites. We must wait for more accurate knowledge of the physiology and pharmacology of the glands and for more certain means of estimating disturbances before we shall be able to say definitely that a given psychoneurosis is the result of this or that glandular syndrome.

WECHSLER, New York.

TETANY IN THE EUNUCHOID: REPORT OF A CASE. H. W. WOLTMAN, J. Nerv. & Ment. Dis. **50**:433 (Nov.) 1919.

Woltman reviews briefly the literature on tetany, and reports a case because it has certain unusual features and because it is related to endocrine disorders of another source. The family history revealed convulsions and laryngospasm in various members of the family during childhood. A history of migraine was obtained and one brother developed a condition which strongly suggested an angioneurotic edema. The patient, aged 36, a barber, had had general convulsions beginning at the age of 1 and disappearing spontaneously at the age of 2. From the fourteenth to the sixteenth year he had had inflammation of the bowels associated with vomiting. At 21 a herniotomy and appendectomy were performed; after this he was perfectly well for a period of eight years. He then married. His wife has never been pregnant. Seven years prior to his application to Dr. Woltman for examination he noticed that his legs became easily tired; this condition persisted until one day his legs jerked up like a horse with "spring-halt." In the fall of 1915 the spasms appeared with increasing intensity, frequently five or six times a day. He had attacks which caused his mouth to remain wide open from thirty to forty minutes at a time; on other occasions trismus was present. The convulsions diminished in frequency, and in January, 1916, the patient was able to get about with the aid of crutches. His legs then began to swell and in August, 1916, they were two or three times their normal size. He also had edema of the abdominal wall and the back, and probably some ascites. In September, 1916, he suddenly cried out, lost consciousness and remained unconscious for three days. His legs became flexed at an angle of about 45 degrees and remained in this position. He lost weight very rapidly, so that one's fingers could be placed around his thigh; his face had a puffed-out appearance, and he was almost completely bald. The skin was dry and scaly "like a fish." The right testicle on examination was found to be flabby and small; the left was absent as the result of an orchidectomy which had been performed for tumor.

Woltman describes certain findings in his examination which he does not adequately explain on the basis of a tetany or other pluriglandular disturbance, and a report of postmortem is missing. Among these findings are a Babinski reflex, Chaddock's sign, a Rossolimo reflex and an ankle clonus on the right side, and a pallor of the disks suggesting a primary optic atrophy. He describes, in the course of the patient's illness, a number of attacks of tetany. Medication was of no avail, and the patient died March 3, 1917, greatly emaciated.

The author reviews some of the clinical features of tetany and points out the endeavor to identify more definitely the chemical nature of the substance at work, referring to Fuch's reference to ergotism, and to Eppinger's reference to histidin chlorhydrate. He calls attention to the work of MacCullum and

Voegtlin in relation to calcium deficiency. He quotes the views of Wilson and others relative to the ammonia content of the blood, and reviews Koch's theory.

He interprets the abnormal dryness and scaling of the skin in his case as suggesting thyroidal features, the presence of pigmentation noted during one of the exacerbations as of possible suprarenal origin and, finally, the direct evidence of disturbance of the gonads in the development of a testicular tumor with the clinical picture designated variously as pseudohermaphroditism, eunuchoidism, etc.

Pollock, Chicago.

CONTRIBUTION A L'ETUDE DE LA PATHOGENIE DU TABES.

Odilon Gallotti and Sergio Azevedo, Rev. neurol. **26**:489 (June) 1919.

The pathology of tabes is one of the most frequently discussed problems in neuropathology. Advances are being made, however, in spite of disagreement of opinion; Schaudinn having conceived the idea that sleeping sickness is caused by a form of spirochete, Spielmeyer goes a step farther, contending that since syphilis is a spirochetosis, there must be some close relationship between sleeping sickness and general paresis of the insane. This in turn has led him in his studies to the opinion that tabes due to the trypanosome (in animals) is identical with syphilitic tabes in man. He founds this opinion on his experiments with dogs into whose subarachnoid spaces he injected the trypanosomes. By the Marchi method, eighteen out of forty-two animals showed degeneration of the posterior spinal roots, of the optic nerve and of the sensory branch of the trigeminus. Clinically, however, the animals showed no symptoms other than diminished reflexes of the fore legs.

The degenerative processes seem to show a preference for the cervical segments of the cord. The lesions in the peripheral nerves, however, are not to be distinguished from those produced by lead, alcohol or pellagra.

The chief resemblance, pathologically, between trypanosomic tabes and that of man is that both choose the focus where the posterior root pierces the pia mater. Spielmeyer's experiments on dogs prove conclusively the correctness of Schaffner's earlier contention that the cord lesion is secondary to the radicular lesion.

Spielmeyer's results led the writers to experiment on three dogs. Not being able to obtain a special strain of *Trypanosoma brucei,* they had recourse to *Trypanosoma gambiense* and *T. cruzi* which seem to have a special affinity for the nervous system. The writers describe their technic of inoculation. Two of the three dogs died; twenty-five hours later they found the trypanosoma in the blood of the animal that survived the experiment. At the end of three months they killed this animal, removed the brain and cord, stained sections in Mueller's fluid and by the Marchi method; nothing startling being discovered, they tried the Weigert method and found advanced signs of nerve degeneration in the lumbar region. They found, particularly, fibers that had lost the myelin sheath. In the cervical region, they found occasional lesions in the anterolateral column and constant changes on either side of the posterior root zone. Sections of the dorsal cord showed small lesions.

They conclude with the statement that their work agrees in its results with that of Spielmeyer.

Jones, Detroit.

THE PATHOLOGICAL CHANGES IN THE CEREBELLUM IN ACUTE ANTERIOR POLIOMYELITIS. Hubert S. Howe, Neurol. Bull. **2**:261 (July) 1919.

From the review of the literature, Howe found that little attention has been paid to pathologic changes in the cerebellum. Clinically the diagnosis of cerebellar affection is usually diagnosed by the ataxia, but as others have pointed out and as Howe, himself, says, there are many other locations in the cord and brain which may account for the ataxia. The author cites a few case histories reported by other observers to show the clinical history of poliomyelitis with cerebellar changes, but one of the cases reminds one more definitely of a cerebellar apoplexy than of an encephalitic process.

Howe quotes many authors to show that pathologic changes in the cerebellum are absent in poliomyelitis, are usually not marked, and are often difficult to find when present. Some authors have described mild perivascular infiltration and changes below the granular and Purkinje cell layers; others described the lesions as thrombosis of the terminal arterioles; still others found pial lymphocytic infiltration and slight hemorrhages.

Howe's findings are: Macroscopically, the cerebellum appears edematous and congested. Microscopically, the cerebellar pia frequently shows diffuse inflammatory changes, more marked than in the cerebral pia and closely resembling that of the cord. Edema, hemorrhage, congested vessels and exudated cells separate its layers, but the picture differs from ordinary meningitis in that there is no fibrin and no exudate on the pial surface. The infiltrating cells are lymphocytes, few large mononuclears, polyblasts and polymorphonuclear leukocytes. The vessels are congested, but no thrombosis was ever encountered. The most striking change is cellular infiltration in the Virchow-Robin lymph spaces, which, however, is less common than in the cord and also differs from the latter in that the white matter presents the most frequent sites.

The ectodermic changes occur both in the white and gray matter of the cerebellum. Focal areas of cellular accumulation are seen, which are less intense than in the gray of the cord, and do not seem to bear any relations to blood vessels or nerve cells. The Purkinje cells in the infiltrated areas only show degenerative changes. As in the cord, the infiltrating cells are made up of lymphocytes, polyblasts, ameboid neurologic cells, plasma cells and a few polymorphonuclears. The Purkinje cells may show slight cloudy swelling but are usually normal. The changes in the white matter are more marked than in the gray, and affect mostly the deeper white substance of the peduncles and the central white.

The author concludes that, inasmuch as the poliomyelitis virus has little affinity for the cerebellar nerve elements and the changes are mostly confined to the white substance, "the clinical symptoms referable to the cerebellum are in reality due to interference with the function of its connecting pathways and not to the destruction of nerve elements."

Wechsler, New York.

A CONTRIBUTION TO THE STUDY OF THE PATHOLOGY OF HUMAN AND EXPERIMENTAL POLIOMYELITIS, BASED ON CASES OCCURRING DURING THE EPIDEMIC OF 1916 IN NEW YORK CITY. Hubert S. Howe, A.M., M.D., J. Nerv. & Ment. Dis. **50**:409 (November) 1919, continued from **48**:235.

Hubert S. Howe continues with the protocols of cases of experimental poliomyelitis, and discusses his findings which he summarizes as follows:

1. The tissue changes in acute anterior poliomyelitis are of three distinct types—those which predominantly affect the ectodermogenic elements, those which predominantly affect the mesodermogenic elements, and those which affect both these elements about equally.

2. The ectodermogenic type of tissue change in the central nervous system of man is polymorphous; that is to say, the cellular constituents derived from the ectoderm show no one form of degenerative process. The reaction in the ganglionic cells and neuroglia makes it possible to recognize no less than eight different forms in the degenerative process consequent to the poliomyelitis infection.

3. The degenerative changes in the ectodermogenic elements are so diverse as to make it impossible to conceive of them as phases in a single disintegrative process.

4. The mesodermogenic type of tissue reaction presents the more general reaction of the organism to the infection and manifests itself by uniform changes affecting not only the central nervous system, but also the lymphoid tissues throughout the body.

5. Changes in the elements derived from the ectoderm, neuroglia and ganglionic cells are, in the main, not secondary to or dependent on the changes in the mesodermogenic tissues, but represent a specificity on the part of the ectodermogenic elements in their reaction to the virus.

6. The susceptibility of the motor cells in general and the anterior horn cells, or their homologues in the brain stem, in particular, furnishes further evidence of the specificity of the poliomyelitis virus.

7. The possibility of such definite specificity in a given virus is demonstrated by the exclusively ectodermogenic reaction observed in the pathologic changes resulting from the feline epidemic which was coincident with the poliomyelitis epidemic of 1916.

8. While it is possible to conceive of certain conditions which might underlie a selective reaction of such a specificity, the determination of these conditions still remains to be disclosed by future work in this field.

9. In human poliomyelitis, the pathologic reaction to the virus is a mixed one, in which both mesodermogenic and ectodermogenic changes occur. It is undoubtedly true that the most severe impairment of function results from the changes in the elements of ectodermal origin, while it seems probable that the more transitory or even abortive disturbances of normal function are the result of changes almost, if not wholly, limited to mesodermogenic tissues.

Pollock, Chicago.

EARLY NEUROSYPHILIS ASYMPTOMATICA WITH REPORT OF OBSERVATIONS AND CASES. Joseph Victor Klauder, M.D., Am. J. Syphilis **3**:513 (Oct.) 1919.

The author expresses the belief that probably in all cases of primary and secondary syphilis the nervous system is invaded by the treponema. In support of this view he has collected from the literature and from his own observations clinical and laboratory evidence.

Spinal fluid studies on 214 cases of primary syphilis by various syphilologists are cited, only forty-seven of which showed an abnormal spinal fluid content. It is to be noted that the abnormality usually consisted of a slight increase in the cell content and that there was a wide difference of opinion as to what constituted a normal cell count. Only two cases showed a positive spinal fluid

Wassermann reaction. The percentage of neuraxial involvement found in secondary syphilis is given as from 10 to 90 per cent., the number of cases studied not being stated.

Treponema pallidum was demonstrated in the spinal fluid in fourteen cases of primary and secondary syphilis; the spinal fluid in these cases was otherwise frequently normal; the subjective and objective phenomena were frequently absent.

The clinical manifestations of early neurosyphilis are reviewed, the symptoms virtually always being referable to an involvement of the meninges. To the usual three clinical types of meningeal involvement he adds a fourth called the asymptomatic group, which as the name indicates, lacks subjective or objective symptoms; but it possesses conspicuous spinal fluid evidence.

Of the cranial nerves, the eighth is the most frequently involved. The others involved, in order of frequency, are second, seventh, third, fourth, fifth and sixth.

In a series of twenty-five cases the author made an initial examination of the spinal fluid in the untreated eruptive stage of syphilis and a second one after the administration of four intravenous injections of arsphenamin 0.5 gm. at from seven to ten day intervals, this being the only treatment given. In every case there was an accentuation of the eruption, and a Herxheimer reaction after the first treatment. In one case a negative spinal fluid became positive after treatment; in another, in addition to spinal fluid changes, clinical evidence of neurosyphilis in the form of a severe headache developed, but disappeared on further treatment. The latter phenomenon is ordinarily known as neurorecidive. The term neurorecidive and the Herxheimer reaction are defined, and a brief review of the literature on the subject is made.

The author cites his own observations in substantiation of the view expressed that neurorecidive is an expression of the Herxheimer reaction. The neurorecidive phenomenon is divided into two stages—the laboratory or asymptomatic, and the clinical or symptomatic. The former is styled "neurosyphilis provocativa asymptomatica"; the latter, "neurosyphilis provocativa symptomatica."

The Herxheimer reaction is regarded prognostically of unfavorable significance. Its excitation should therefore be avoided in the treatment of syphilis by preceding the arsphenamin therapy with a course of mercury because of its milder spirochetacidal properties.

Just criticism is made of the insufficient and exclusive use of arsphenamin in the early treatment of syphilis.

Haines, Philadelphia.

IRREGULARITY IN A PSYCHOLOGICAL EXAMINATION AS A MEASURE OF MENTAL DETERIORATION.

Sidney L. Pressey, Ph.D., and Luella W. Cole, A.B., J. Abnorm. Psychol. **13**:285 (Dec.) 1918.

Pressey and Cole present a comparative study of the amount of "irregularity" or so-called "scatter" between a group of feebleminded and psychotic subjects all showing mental ages from 8 to 12 years. "Irregularity" is used as a term to denote the sum total of the variations from the original point scale for norms. For instance, the feebleminded score is characterized, not only by its low level, but also by its regularity and the absence of such discrepancies as are brought out in psychotic persons; namely, the inability to pass certain tests, together with the ability of passing others which are respectively far below and above the mental age as determined by the entire scale. The group tested comprised 158 defectives, sixty-seven schizophrenics and fifty-five chronic alcoholics. The average "irregularity" of the defectives totaled 17.5; the schizophrenics 20.1 and the alcoholics 22.3. That there was considerable overlapping is evidenced by the fact that 25 per cent. of the feebleminded revealed as much "scatter" as did the average praecox patient. From an analyses of the results the authors then selected five differential tests. These not only emphasized the preponderance of "irregularity" in the psychotics (6.9 for the schizophrenics and 8.2 for the alcoholics as against 4.7 for the defectives), but also markedly decreased the overlapping. In only 10 per cent. of the members of the feebleminded group was the "scatter" as great as in the praecox subjects, and in only 2 per cent. as compared to the inebriates. "Irregularity" in mental diseases may be determined by a variety of factors, such as temporary negativism, retardation or delusional trends. It therefore measures more than merely the amount of mental deterioration. A point of some practical importance lies in the suggestion that pre-existing and basic intelligence deficiency may be suspected in those psychotics who test low but evenly, i. e., without "irregularity."

The authors realize that it would not be justifiable to draw far-reaching conclusions from the limited number of subjects tested. Furthermore, there are tremendous obstacles that stand in the way of applying a pure intelligence scale to abnormal mental states. It is somewhat doubtful if the various factors in the psychoses which might give rise to "irregularity" can ever be standardized. Even in the so-called deteriorating psychoses, such factors must vary greatly from time to time and in the same patient. For instance, in a group of praecox patients one may expect much individual variation in the affective sphere, in the domain of the will and in the ideational realm within comparatively short periods of time so that the index of "irregularity" will be found to be far from constant, even in the same subject.

Strecker, Philadelphia.

A CASE OF TUMOR OF THE CORPUS CALLOSUM AND FRONTAL LOBES. C. C. Beling, M.D., and H. S. Martland, M.D., J. Nerv. & Ment. Dis. **50**:425 (November) 1919.

Beling and Martland report a case of a male, white, 54 years of age who, at 30 years of age, suffered for a few days from vertigo. Jan. 23, 1914, some hypersensitiveness to light and diminished vision were noted. In April, 1916, considerable loss of memory was revealed, and May 19, 1916, he had a sudden convulsive attack which began with twitching of the right arm, hand and leg. May 24, 1916, he had recovered sufficiently to resume his work. A week or two later he was talking and acting in a silly manner. Examination revealed a bilateral optic neuritis, and the gait was unsteady and somewhat ataxic; "clonic perseveration" (Liepmann) showed markedly in his handwriting. Involuntary urination and defecation were present. Because of the disturbances of association resulting in apraxia, chiefly of an agnostic, ideomotor, aphasic and agraphic character, a clinical diagnosis of tumor of the corpus callosum and frontal lobes was made. Automatism and defects of volition (lack of initiation of movement ideas) were explained by an involvement of the left prefrontal lobe. A postmortem examination made eight hours after death, on July 3, 1916, revealed a tumor occupying the inner halves of both frontal lobes, the genu and the anterior one-third of the body of the corpus callosum. The tumor was infiltrated in character, fairly well circumscribed but not encapsulated. It presented the gross characteristic of a telangiectatic, infiltrative, noncircumscribed glioblastoma. It was composed of proliferating neuroglia cells presenting a whirl-like, radiating and wavy arrangement. The greater part of the tumor presented extensive retrograde changes, especially necrosis and hemorrhage.

Pollock, Chicago.

SCIATICA WITH MUSCLE HYPERTROPHY. Capt. C. Pastine (Chief Neurological Service, Field Hospital 227), Riv. di patol. nerv. **24**:46, 1919.

The author says that Roccavella added eight new signs of sciatic neuritis to the twenty-five already known as a result of observation of many cases during the war. The author adds still another, namely, hypertrophy of muscles instead of the usual atrophy seen in sciatica. Careful reading of the ten case histories presented shows that all of them are open to serious criticisms as to diagnosis. No sensory examinations are detailed; the neurologic signs are scant. Achilles' reflex is present and the patellars are active in several and clonus in one. The author speaks of tenderness and pain in the gluteal, and thigh and calf hypertrophies. Many of the ten patients had abnormal attitudes caused by spinal disability or deformity or compensatory scoliosis from hip and knee joint involvement. The author's finding of hypertrophy of muscles in sciatic neuritis is seriously questioned. During my service as neuropsychiatrist to the American forces in Italy during the war, I was ordered on detached service to the author's military hospital and directed the neurologic service during the author's illness. I saw many of the cases of "sciatica" so-called. No sensory examination worthy of the name was recorded. The hospital did not boast of a roentgen-ray apparatus. Many of the cases were gluteal or other types of myositis; many were surely more or less obscure joint and spine conditions. A few that I saw were true sciatic neuritis. None of these had hypertrophy of muscle. One case of "sciatica" was a tabetic. Of the ninety-eight cases of "sciatic neuritis" seen by the author, ten presented muscle hypertrophy. All of these conditions came on after prolonged exposure to rain and snow and cold in the mountain trenches which this hospital drained. The author admits that many of the cases showed cyanoses, thermic and vasomotor symptoms in the extremities, accompanying so-called "trenchfoot" and not sciatic neuritis. The author's statement that true sciatica is accompanied in many cases by muscular hypertrophy directly dependent on the neuritis is open to grave doubt.

Osnato, New York.

AN ACUTE PRISON NEUROSIS OF THE ANXIETY TYPE. N. S. Yawger, M.D., J. Nerv. & Ment. Dis. **50**:319 (October) 1919.

N. S. Yawger analyzes this type of neurosis in a brief and comprehensive manner. He states that it is more common in penitentiaries to which, generally speaking, felons are sentenced, than in the smaller jails where those only guilty of misdemeanors are interned. About one third of the convicts coming up for pardon show nervousness to a greater or less extent and in one tenth of these offenders the subjective symptoms are so suggestive as to be almost diagnostic. Although hoping for pardon is productive of a more intense neurosis than is the approach of the expiration of a minimum sentence, on obtaining freedom the pardoned person recovers more promptly. The subjective evidence of the disorder appears in the form of restlessness, decreased power of mental concentration, irritability, hypochondriac and introspective manifestations, dreams of a disturbing nature, often broken by distressing insomnia, dyspepsia, genito-urinary manifestations, cardiac irregularity or respiratory disturbances; usually, there is an appreciable loss in weight and in smokers, an increased use of tobacco.

In the consideration of the nature of this disorder the author believes that undoubtedly the reactions of this anxious period are the result of important factors aside from the sexual sphere. Such factors include the reactions to a situation wherein the prisoner knows that detainers have been lodged against him and that he will be homeless, and the certain knowledge that he will be an unwelcome member of society.

Pollock, Chicago.

CONCERNING HAMLET AND ORESTES. John T. MacCurdy, M.D., J. Abnorm. Psychol. **13**:250 (Dec.) 1918.

Must "autistic thinking" follow certain channels that are common to all individuals, no matter how far separated in time, place, or civilization?

Hamlet and Orestes figure in stories derived from sagas of the early Northmen and pre-Greeks. And yet there are striking likenesses in the plots of their stories and in the character and "madness" of the heroes. The type of insanity is given as dementia praecox because the following symptoms appeared: seclusiveness, hallucinosis, ideas of reference, scattered speech, inappropriate affect, filthiness, and "queer behavior with sudden, unexplainable assaultiveness." With these symptoms are linked ideas of incest and death. The author's conclusion is that the inspired dramatist in dealing with a hero of incestuous tendencies, is forced by his very genius to paint about his hero the clinical picture of dementia praecox, for in all men there is a "basic, unconscious connection between ideas of a given type" and this disease.

It is too much to ask that the big idea which underlies this article and makes it stimulating shall carry the assumption that there is no dementia praecox without an Oedipus complex or to depend on a definite diagnosis of the disease of a mythical hero who is Shakespeare's Hamlet mixed with many Norse pre-Hamlets.

Bond, Philadelphia.

A NEW CONCEPTION OF EPILEPSY. P. Hartenberg, Presse méd. **27**: 664 (Nov. 8) 1919.

The author offers a new conception of epilepsy. He gives as the cause of the disease *inhibition* of the centers of the cortex rather than stimulation. As proof he cites the frequency of brief amnesia and vertigo; in other words, the petit mal compared to grand mal. These are cessations of functions rather than increase of them. He goes further—he says that grand mal is only the petit mal plus convulsions plus coma. His formula is: amnesia = unconsciousness; vertigo = unconsciousness plus falling; grand mal attack = unconsciousness, plus falling, plus convulsions, plus coma. He asks whether convulsions may not also be the result and product of inhibition, and cites the numerous comas which are accompanied by convulsions. In short, inhibitions of the higher cortical centers may set free muscular discharges. Only two conditions are necessary, and these are both present in epilepsy, namely, that the inhibition be brusque and that it be profound. The analogy of the convulsive movements to those produced by a lesion of the corticothalamic tracts (forced laughing and crying) stimulates Hartenberg to believe that the convulsions of epilepsy are due to absence of cortical control of the lower centers.

By experimenting with the effect of strychnin, Dr. Hartenberg found that it notably diminished the symptoms rather than increased them, which lends plausibility to his theory.

In short, epilepsy is not a convulsion, but a state of unconsciousness. It is due to inhibition of the activities of the upper cortical centers rather than to irritation of the motor centers.

This article is interesting and well worth a careful perusal.

Kraus, New York.

THE INSIGNIFICANCE OF LOCAL DISTURBANCES IN THE PRODUCTION OF CONSTITUTIONAL NERVOUS SYMPTOMS.

Charles McKendree, Neurol. Bull. **2**:253 (July) 1919.

The author bases his conclusions on his observation of some 3,500 neurologic cases, the vast majority of which were so-called functional cases. Bearing in mind the great emphasis that is laid on focal infections, reflex irritation, visceral disturbances, etc., which have been so frequently incriminated as the causes of obscure psychic complaints, he tried to study a number of cases. Wherever there appeared the slightest indications, he referred the patients to the dentist, oculist, nose and throat specialist or gynecologist, etc. Frequently each specialist found something which he was sure was the cause of the headache, the pain or indigestion. The patients underwent palliative, corrective or radically operative treatment, but despite that their chief symptoms remained. He quotes five cases to illustrate the futility of treatment in patients who showed no general physical or neurologic signs, but who were supposed to suffer from some obscure "cause."

Without trying to minimize the importance of special causes in the production of general symptoms, McKendree revolts against the faddist tendency in medicine to run away with oneself as focal infectionists, for instance, do when they try to explain every vague symptom, or any condition from bunion to brain tumor, by an apical abscess or ungeometrical tonsillar crypt. He agrees that there are cases, here and there, which benefit from special attention, but those cases show definite symptoms which can be well correlated with the underlying causes. The vast majority of constitutional nervous symptoms, however, have no such etiology, and the few who do show improvement with special attention would show it just as well on general hygienic or other treatment. Besides, most of the patients do not respond to such treatment, and what is more important, they become confirmed neurotics because of the radical attempts at cure. The author, therefore, pleads for a little more common sense in the evaluation of symptoms and the bridling of the radical tendency to seize on insignificant and unsubstantiated conditions as the root of all constitutional nervous disorders.

Wechsler, New York.

THE CEREBROSPINAL FLUID IN HERPES ZOSTER, AND THE RELATION OF HERPES ZOSTER TO SYPHILIS.

W. H. Brown, M.D., Captain, R.A.M.C., and Dr. B. Dujardin, Brain **42**:86 (April) 1919.

The authors report the cerebrospinal findings in twenty-five cases occurring in Captain Brown's experience, and sixteen cases observed by Dr. Dujardin. The cytologic estimations showed striking, inconstant and puzzling results. In the majority of cases, twenty-eight, there was some degree of lymphocytosis, but this varied enormously from normal to an intense lymphocytosis (470 per cubic millimeter). In ten cases the counts registered over 50 cells per cubic millimeter, in ten, the counts registered within normal limits. In the majority of cases there was a slight increase of albumin. In only one was there a clear, decided ring obtained by the Nonne-Apelt method of testing for globulin.

Relative to Dr. Dujardin's statistics, the striking feature is the frequent incidence of herpes zoster among syphilitic subjects. Of 1,200 cases of syphilis in one stage or another, nine developed herpes zoster. Among 13,000 other patients only ten cases of herpes zoster was seen. The authors state that it would appear that a predisposition to herpes zoster existed in syphilitic subjects. They also point out the fact that in their cases herpes zoster in syphilitic subjects had a predilection for the lumbar and sacral ganglions. Conversely, in a case of herpes zoster the possibility of a prior syphilitic infection must be considered, more especially if the herpes involves the lower spinal ganglions.

POLLOCK, Chicago.

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9. NERVOUS AND MENTAL DISEASE IN UNITED STATES TROOPS. Pearce Bailey, M.D., New York.

Statistical tables showing incidence as regards age, locality, social condition, nationality, length of service, methods of detection, etc.

DISCUSSION

Dr. Joseph Collins, New York: I should like to speak of two points that were brought out in the paper by Dr. Bailey. The first is: There are in the United States a great number of potential nervous and mental diseases among the youngest and most active persons physically of the community. They are not recognized by the medical profession, which includes the neurologist. Therefore there must be something radically wrong with the relationship of the medical profession to the community. The second point is: There are in the community a large number of young men capable of earning their livelihood who yet must be classified as high grade imbeciles. So far I have not learned much from the war concerning the pathogenesis of nervous disease, but I have learned something concerning my relationship to the community and have experienced a decided quickening of my communal conscience. We have a larger work cut out for us than the diagnosis, interpretation and attempt at treatment of well developed disease, and that work lies in the field of cooperative medical pedagogy and preventive medicine. The medical profession of this country was unprepared to make the so-called neuropsychiatric examinations when war was declared, unprepared without intensive training to take care of neuropsychiatric cases when they were detected and when they resulted from war and the incidences of war. It is now less prepared than many nations to enter into the work of reconstruction from the neuropsychiatric point of view. The cause of those defections may be summarized in one sentence. The neurologist has never been properly taught in this country and the internist has devoted his energy to scorning the neurologist and treating with contumely the things he must now recognize. It is our duty as neurologists and psychiatrists to lead medical thought in this direction. We must convince the profession and the community that it is the man as well as the disease that must be treated; that the emotions which are an integral part of a man are bound up as closely through the endocrine system through the somatic nerves with disease as man is bound up in original sin. We must embrace our opportunity now to do missionary work, not among the uneducated but among the leaders of medical thought and endeavor in this country: first, by showing that the nervous system and its disorders is not a sterile field of medical research and labor; secondly, by making men capable of teaching neurology and psychiatry; thirdly, by not permitting a student who has not acquired the principles of neurology and psychiatry to enter the portals of medicine, and fourthly, by recognizing that it is more important that a favorable medical atmosphere be developed than that hospitals for nervous diseases be built. It is our duty and our opportunity to develop that atmosphere.

10. SOME PSYCHIATRIC LESSONS OF THE WAR. Dr. Thomas W. Salmon, New York.

No discussion.

11. HYSTERIA IN THE LIGHT OF THE EXPERIENCE OF WAR. Lieut.-Col. Arthur F. Hurst, M.D., R. A. M. C., London, England.

This article appeared in the November issue of the Archives of Neurology and Psychiatry, page 562.

DISCUSSION

Dr. Morton Prince, Boston: I would like to say a few words in connection with Colonel Hurst's paper, and to pay a tribute to his great skill in curing his patients. But when he comes to the pathology that he has adopted from Babinski's teachings I am compelled to take issue with him. If it is said that hysteria is due to a psychic cause, we can all agree, for that is a well accepted fact, but when he says that hysteria is solely the result of suggestion, the conception in my view, is entirely inadequate and is based on a consideration of not only a limited number of types of hysteria but of a limited number of its manifestations and without regard to the fundamental underlying pathologic state. As the "accidents" or manifestations of hysteria Babinski recognizes only those phenomena which he avers are capable of being reproduced experimentally by suggestion and correspondingly can be made to disappear by suggestion. The essential symptoms that he recognizes as hysterical are: convulsive attacks, paralyses, contractures, tremors, choreic movements, troubles of phonation, respiration, sensibility and sensorial troubles. He defines hysteria in the following way: "Hysteria is a *pathological state* manifested through troubles which it is possible to reproduce by suggestion in certain subjects with a perfect exactness and which are susceptible of disappearing under the influence of persuasion (contra suggestion) alone."

Strange to say, however, after the formulation of this definition, we hear nothing more of the pathologic state—only an elaborate and clever exposition of its manifestations. This failure to keep in mind, on the part of Babinski, the conception of the pathologic state is the weak point in the edifice which he builds up. Dwelling only on symptoms, he fails to grasp the essential problem of hysteria, losing sight of the pathologic state which should be the goal or solution sought. He confines himself to certain physiologic phenomena and loses sight of the fact that many of the so-called troubles which are the manifestations of the "hysterical state" are pure mental stigmas. These are not included in his first group of pithiatic symptoms, i. e., they are not recognized as hysterical, though they can be induced in favorable subjects by suggestion. Necessarily, therefore, he fails to recognize that many of the cases which he makes use of to support his thesis were already in the "hysterical state" and manifested classic manifestations of hysteria. The cases that had been exposed to emotional trauma, he supposes, were free from hysteria until they were later the victims of suggestion. The consequence of this lack of vision is that when he comes to his data, to the cases which he cites to show that hysteria is not induced by emotion but only by suggestion, he naively describes cases in which every clinician ought to recognize that the patient had already developed hysteria and had already exhibited classic hysterical symptoms of a mental sort induced by emotional trauma or mental stress and strain, before suggestion, even in his own opinion, had had effect.

The corner stone of Babinski's thesis, then, is that the hysterical accidents (pithiatism), very limited according to his conception as we have seen, are always the result of suggestion—autosuggestion or heterosuggestion.

I have no cause to quarrel with Babinski for insisting that hysterical phenomena can be suggested. Every neurologist has seen often enough examples of this in his practice, and, indeed, is aware that he himself has suggested such phenomena, intentionally or unintentionally. Nor can any one doubt that a large proportion of hysterical stigmas, particularly those following traumatisms, have been so suggested, generally unconsciously, by the examining physician. But that all have been so suggested, or what proportion have been so suggested, is another question. Given a certain abnormal state of mind, symptoms can be manufactured almost ad libitum. In such a state the symptoms can be educated, just as by reeducation they can be made to disappear.

But what is the underlying pathologic state that permits such suggestibility, and which Babinski passes over? And is this "state" induced by suggestion alone? This is the real problem. Every one recognizes at the present time that the "pathological state" of hysteria is an emotional dissociation of one or more of the normal integrated psychoneurologic systems manifesting itself by loss of certain mental and neurologic functions, on the one hand, and overactivity of certain other functioning systems, on the other. If you do not like the psychologic term "dissociation," you may use the physiologic term disintegration, or inhibition or repression. We are not now concerned with the mechanisms by which these phenomena are brought about. The question, then, becomes not simply whether the manifestations are due to suggestion and to suggestion alone, but whether the dissociation or disintegration is induced by suggestion and suggestion alone. Babinski devotes much effort to showing that emotion, contrary to the classical view, does not and cannot induce hysteria. He even goes so far as to maintain that "when the human soul is shaken by sincere, profound emotion, there is no longer a place for hysteria." If emotion, then, can be eliminated, no other agent, he assumes, is left but suggestion. In support of this thesis he contends that hysterical phenomena never appear at the moment of or immediately after the emotional shock, when the emotion is at is height, but that always "between the emotional shock and the presence of hysterical ("pithiatic") accidents there is an intervening phase, sometimes quite long, which Charcot called the "phase of meditation," during which autosuggestion or heterosuggestion have the opportunity to intervene and induce the accidents. As evidence he cites the observations of numerous writers who had the opportunity in this war to observe so-called "shell shock" at the front and behind the lines. Babinski seems at first to be supported by these observations, for it appears that those symptoms of hysteria which Babinski elects to call alone pithiatic or hysterical—that is the paralyses, anesthesias, etc.—rarely developed at the time of the emotional shock, but appeared after an interval, when the soldier had reached some place behind the lines.

A study of the reports, however, shows that the cases, apparently without exception, exhibited at the emotional period immediately following the shock, hysterical mental symptoms of a very marked character. Among the symptoms commonly described are: amnesias, hallucinations, deliria, inability to respond to questions, even when attempts were made to force a response, apparent incapacity to perform a voluntary act, states of hebetude, stupor, confusional states, states allied to fugues, mental dulness, irrational states, tachycardia, tachypnea and tremor.

What are these, it may be asked, with the exception of the three last (which are only the physiologic manifestations of emotion), but states of dissociation with automatisms or unregulated and uncontrolled functioning of disintegrated psychologic systems? They are, from the modern point of view, typical and pure symptoms of the hysterical pathologic state. Amnesia, for instance, as already pointed out, is well recognized as a manifestation of dissociation. In these very cases, then, cited by Babinski in support of his thesis, we have evidence of hysterical manifestations induced by something else than suggestion, and we must remember that this cause is conditioned, at least, by emotion. It may also be pointed out in passing that these mental symptoms, following the discharge, may be brought within Babinski's own definition of hysteria in that they are capable of being produced experimentally by suggestion.

That certain symptoms like paralysis, anesthesia, dumbness, deafness, etc., do not appear until a later period may be a fact, as a matter of observation, but the real question is *why* do these particular symptoms appear later, while other and mental symptoms appear at the height of the emotional discharge? It is a question of the *why*. The fact that seems to have been brought out by observation during this war is that the hysterical state, manifesting itself by mental dissociation, can be induced immediately under mental stress and strain at the moment of the emotional discharge, while certain other symptoms, in the great majority of cases, develop only after a period of incubation.

Babinski seems to assume that because this latter class of symptoms develops only after a period of incubation they must be due to suggestion, particularly to heterosuggestion, although autosuggestion may be the genetic factor. To this assumption exception may be taken. That many hysterical stigmas—particularly the paralyses anesthesias, crises, deafness, numbness, etc. —have their genesis in psychologic factors, there can be no doubt, and I think that since 1885 there never has been any doubt. That we have all known for a long time. But this is far from saying that they are due to suggestion, whether autosuggestion or heterosuggestion. The genesis and the mechanism are far too complex for such a simple explanation. They may be called "defense reactions," if you like, according to one theory; but the psychologic mechanism of a defense reaction is complicated. This may be due in individual cases to mental conflicts, to subconscious mechanisms of different kinds, etc.

Babinski's fundamental error is in not recognizing that the hysterical state is one of functional dissociation and that any psychologic factor capable of producing such a dissociation, whether it be emotion or a conflict, is capable of producing hysteria. The mechanism by which individual symptoms is produced is another problem. It may be suggestion, as we all know, or it may be a very complicated mechanism which still requires solution.

Dr. Foster Kennedy, New York: Hysteria has been described as a dissociation of the personality, and Colonel Hurst has portrayed that dissociation. But he must have appreciated that such dissociated personalities were unstable, and I should like to have him say a few words about the disposal of such soldiers after cure—whether an attempt was made to educate each man to be a more stable personality, or whether disposal was made of him so that he was put in an environment where easy adaptation was made possible for him. Without giving this information, Colonel Hurst is apt to give too easy a solution of the problem. I am in entire agreement with him regarding the functional, emotional and "suggested" character of the so-called main gêlé described by Babinski as being produced by reflex neural physiologic block consequent on trauma. I came to the conclusion that probably every wound of the upper extremity was associated with a temporary defensive immobilization; the man feared to move his injured arm, and if he were asked if he could do so he would invariably reply in the negative. If the medical officer took such a statement at its face value, the seeds of hysteria were immediately sown. The improper suggestions which colored these pictures and produced the paralysis were almost always the result of incomplete medical knowledge on the part of the medical officer.

Dr. D. J. McCarthy, Philadelphia: I might say, from the standpoint of the estimation of war risk, that this paper is of great value, and I agree with Colonel Hurst that the vast majority of the cases which he describes are purely hysterical. There is nothing new in any of the cases shown, and those of us with war experience during the last four years, could easily duplicate this group.

I am not willing to agree with the statement that this group of cases entirely eliminates the Babinski reflex group. In 1915, I saw some cases that appeared at first glance to be functional, but which proved, on further study, to have an added element not altogether organic—functional in a different sense from the purely hysterical. The reflexes were sometimes active, sometimes lost. The atrophy was sometimes of rapid development and associated with edema. This was particularly true of injuries near the shoulder joint and of the shoulder girdle. It was more common in pulverization of the humerus. This led me to examine a series of cases at the American Ambulance at Neuilly in 1915, immediately after they had received extensive injuries to the humerus, shoulder joint, etc. I found that there was sufficient evidence to indicate a reaction of the peripheral neuron, and its spinal segment, as shown in motor, trophic and reflex manifestations, to lead me to put the other cases into a separate group to which I gave the name of "metameric functional states." Even after recovery these patients present a condition of reflex disturbance, usually in the nature of increased reflex activity with easy fatigability, more marked in the affected side but often bilateral in distribution. Since 1915 I have seen these cases both in army and civil practice. To my mind they present a clear cut and distinct group, somewhat different from the Babinski group, but more limited and better defined. They can be explained by the reaction at a distance of the spinal segments of the cervical cord directly concerned with the sensory supply to the muscle groups, having their origin and insertion on the bone extensively injured.

There is another phase of the subject of great interest to me—the relation of war risk insurance to nerve injuries. In a majority of the peripheral nerve cases studied, a series of symptoms was added to the organic group, to which I gave the name "functional overflow." These hysterical or functional symptoms varied in extent, and could easily be differentiated from the organic symptoms. I may say that this functional overflow was seen not only in the nerve cases, but was as evident and as equally frequent, in the heart cases, gas cases and other visceral diseases. The internist, with his lack of interest in nervous diagnosis, usually overlooked this functional overflow to the detriment of the patient and of the army. One of the most important functions assigned to the staff in the institute at Vichy was to strip the incoming patient of functional or overflow symptoms, before these symptoms became fixed, and to chart the residual organic symptoms for the benefit of the surgeons in the United States, whose duty it might be to operate, or to discharge the patient to civil life, with a pension based on percentage disability. Some highly efficient person saw fit to strip every field card of these valuable records at the port of embarkation for some reason perceptible only to an army type of mind.

Dr. Hugh T. Patrick, Chicago: I want to embrace the psychologic moment to say a word about Colonel Hurst's paper. His presentation was to me an illimitable joy because it was a demonstration of neurologic renaissance in England. At the opening of the war the English were less prepared than any other nation to understand, and consequently to treat, the war neuroses. From Colonel Hurst's presentation and from the reports emanating from his hospital, it is obvious that he has undertaken and is succeeding in a popular propaganda for education of the English medical public in the matter of the psychoneuroses, especially hysteria. That this is, or was, greatly needed is borne out by his categorical statement that nearly all of the orthopedic cases seen by a neurologist proved to be hysterias and that nearly all the military rheumatic cases at Bath were neurologically shown to be the same disorder.

But it is to be added that all of the matter so graphically presented by Colonel Hurst has already been embalmed in medical literature—most of it for many, many years.

Lieut.-Col. Arthur F. Hurst, M.D., R. A. M. C., London, England: I should like to point out, in reply to Dr. Prince, that my definition of hysteria differs from that of Babinski in one important particular. Babinski believes that hysteria is characterized by a pathologic state of mind which manifests itself in an abnormal liability to respond to suggestion, as a result of which symptoms are produced. In contrast to this, I believe that there is no such constant underlying mental condition and that an abnormal degree of suggestibility is merely a predisposing factor. Until actual symptoms have been produced by suggestion, hysteria is not present. I believe that failure to realize this has resulted in a very large number of cases of hysteria remaining unrecognized. My definition has the advantage of embracing all cases which are by common consent regarded as hysteria. Although some such process as dissociation, to which Dr. Prince referred, may take place in many instances, it is certainly not invariably present. Where, for example, is the dissociation in a case of hysterical vomiting, in which the vomiting is a sequel to that orginally produced by the irritation of swallowed gas? The condition is certainly hysterical, as it is produced by suggestion and is curable with extreme rapidity by psychotherapy. Such a man may have been normal in every way before he was gassed, and he is normal in every way after treatment has resulted in the cessation of vomiting. I cannot see where dissociation takes place in such a case. Dr. Prince referred to those cases of Babinski in which the history showed that such an expression of dissociation as amnesia or stupor was present at the onset. Though this was not uncommon in soldiers, a similar history was not obtainable in the majority of cases, and particularly not in those which only developed after they reached the base as the result of a suggestion which was not operative at the front.

Dr. Kennedy asked about the after-history of these patients. We always kept them in the hospital for some time after the removal of their gross hysterical symptoms, so that the effect of hard work could be observed, and any further treatment required by the presence of associated psychasthenia or neurasthenia could be given. In the vast majority of monosymptomatic cases—such as hysterical aphonia, mutism, deafness, vomiting, and contractures—the man was sent back to the firing line after a preliminary period of home service, as it was found that apart from the particular symptom for which he was admitted to the hospital, he was perfectly fit. The subsequent histories showed that these men were not more liable to develop neuroses in the future than other soldiers. On the other hand, patients in whom a psychasthenic element was well marked and in whom there was evidence of hyperadrenalism and hyperthyroidism were rarely sent back to military duty. They were never, however, discharged from the army until they were fit to earn a living in civil life. Patients with so-called cases of shell shock, in whom hysterical symptoms developed as a sequel to an emotional experience, could be sent back to duty when they were treated at advanced casualty stations, but this could be done less often in the cases of patients admitted only after the symptoms had been allowed to become more permanently established by months of over-sympathetic treatment in hospital. Many of these men were given military duty at home, but others who had always had an unstable nervous system were discharged from the service—not, however, until they were fit to earn a living.

In reply to Dr. McCarthy: Our investigation of a very large number of cases of paralysis and contracture of the type described by Babinski as of reflex origin convinced us that at the stage in which they reached us, which was rarely less than three months after the onset and often as long as two or three years, the contracture and paralysis were invariably hysterical and removable by psychotherapy. The associated changes in the mechanical and electrical excitability of the muscles, the alterations in the deep reflexes, and the vasomotor disturbances disappeared instantaneously. The trophic changes, of course, required a much longer period before they disappeared.

12. THE MANAGEMENT OF PSYCHONEUROSIS IN THE CANADIAN ARMY. (Slides and Cinema Pictures.) LIEUT.-COL. COLIN K. RUSSEL, M.D., Montreal, Que.

No discussion.

13. THE MECHANISM OF WAR NEUROSES. DR. SIDNEY I. SCHWAB, St. Louis.

Dr. Schwab gave a definition of war neuroses. He discussed physiologic conservation as an aspect of primitive defense; the primary and secondary instincts; the emotional element; fear reactions; the rôle of polyglandular activation; neuropathic inheritance; predetermining factors; the essential trauma; the mechanism of fixation; the period of automatism, and convalescent conflict.

DISCUSSION

DR. HUGH T. PATRICK, Chicago: I think Dr. Schwab's conception of the psychoneuroses is not only correct but practical and should be carried over into civil practice. I gather that he believes that the psychoneuroses of civil life are not exactly this conservation process which constitutes the mechanism of war neuroses. In many of the civil cases, instead of using the word conservation we might use the word satisfaction, which really amounts to the same thing. The principles of treatment so successfully carried out in the army should be the goal toward which we should strive in all like conditions of civil life, though successful treatment of the latter is much more difficult.

14. THE SYMBOL AS AN ENERGY CONTAINER AND SOME SUGGESTIONS AS TO THE KINETICS OF DISCHARGE. Dr. Smith Ely Jelliffe, New York.

Dr. Jelliffe discusses the human body as an energy system. The inadequacies of the dietary theories as to energy sources are pointed out. He emphasizes the necessity for physiologists to fall back on "vital action" as an adequate explanation for the dynamics of organ action. The necessity for a search for energy sources is not usually considered as such. He discusses the development of speech itself and the kinetics of speech transformations manifested in alterations and evolutions of symbols and as substitutes for things and for conduct. The kinetics of symbolic action as discharged in thinking are also considered. They are illustrated largely by the development and evolution of the word "symbol" itself, which in its own synthesis illustrates the main features of the hypothesis assumed.

DISCUSSION

Dr. Walter Timme, New York: I pass rapidly over the physiologic and dynamic treatment of the subject with simply the statement that I believe Dr. Jelliffe does not use the word energy in the right sense. When Dr. Jelliffe talks of our receptors of "energy" and containers of "energy" he does not mean energy, but energy liberation. Energy is work done along a given path; this work within us may be in the direction of locomotion or the production of heat, or the activity of mental processes. This energy remains latent within us until it is set free—mayhap by physical stimuli, mayhap by symbolic. We produce energy within us with the help of certain agents, whose combination is simply the transmutation from extraneous supplies—such as food, air, sunshine—into easily awakened indigenous compounds. Passing over that, we then come to the various theories regarding the thymus and parathyroid in the production of tetany Uhlenhuth was compelled to advance his theory that the thymus produced a toxin because to him there was no choice left. It had been shown that an overacting thymus, when the parathyroids were normal, produced in an animal a tendency toward tetany and that tetany could be relieved by calcium salts injected into the blood vessels. If in an animal with normal thymus we remove the parathyroids, the same thing occurs, namely, a marked tendency of the animal to progress to tetany, which can again be averted by the feeding of calcium salts. In other words, the thymus produces something which conduces toward tetany in the animal, and which is excreted by means of the parathyroids. Whether or not this can be made applicable in so broad a sense as Dr. Jelliffe has presumably made use of, is left for further thought and more or less critical reading of Dr. Jelliffe's paper when published.

Dr. Smith Ely Jelliffe, New York: Just one word with reference to Dr. Timme's discussion in which I think he tends to prove the points I have tried to bring out: What we know about the transforming power of the chlorophyl system, while as yet very far from complete, does tend to point out that the kinetic vibratory light energy is transformed into a new set of combinations which we call starch, cellulose, etc. The chemical equation is fairly well known although it is not certain what part the manganese plays in the enzyme action, nor are we in precise touch with the photo-electrical processes. That the latter exist is well known. All that I have assumed is that in addition to food there are other types of stimuli or energy sources, which in a manner analogous to the transforming capacity of the chlorophyl system, kindly offered me as an illustration by Dr. Timme, produce responses in the human being and, all taken together, have made man the organism he is, capturing, transforming and releasing these cosmic energy sources.

I then go on to try to indicate that the gradual formation of the symbol is as an energy container and how it is utilized at the intake and the outgo systems of the human beings. The visual purple of the rods and cones is an interesting analogue of the chlorophyl system, but vastly more complicated as they represent an involutionary synthesis of probably millions of years of photoreceptive experiences. This great time element during which the evolution of form as a structuralization of experience has taken place is rarely grasped by any of us, and we are all too prone to accept the impossibly trivial as adequate explanations for the enormous complexities bound up in this evolutionary mechanism. The trivial explanation is our defense reaction to prevent effort to understand these complex dynamics of life. Uhlenhuth says that feeding the larvae of certain newts up to a certain point with thymus will produce tetany-like results; we agree with him, but when he says it is due to a toxin in the thymus, we wait—the toxin hypothesis is so orthodox that we suspect its usefulness. It is so obvious that it does not explain, for what does a toxin do? How does it do it? We are no nearer an explanation of tetany when we say it is due to a thymus toxin, or that it is due, as Paton would say, to disturbance in the guanidin regulation—another type of toxemia. In these explanations the body is considered as a closed system, out of relation to other energy sources than the thymus, or guanidin or what not; whereas it is not a closed system but a closely integrated set of receptors, connectors and effectors which have been undergoing a perfecting process for many millions of years. A reaction like tetany can be understood only as a disturbance in some part or parts of this chain. Is it in the primary receptors, the peripheral arcs, the spinal arcs, the cortical arcs or the final effective muscle arcs? We have tried to show it is due to a disturbance in the calcium metabolism, or that part of it of which the parathyroid is an integral part and the particular function into which the bivalent ration enters is the electrical conductivity at the synapse at the spinal arc. The lowering of the synapse threshold prevents an orderly distribution of the cosmic stimuli, causing a flood—an avalanche. Extensor response, i. e., flight, takes place, or would take place, as is seen in the severe muscle reaction.*

15. SEGMENTAL CEREBRAL MONOPLEGIA. Dr. William G. Spiller, Philadelphia.

The author describes several cases of cerebral monoplegia in which the paralysis was confined to a small segment of a limb. This condition is likely to be confused with nerve lesion or hysteria. Such cases are not numerous in the literature.

DISCUSSION

Dr. Malcolm A. Bliss, St. Louis: This brings to my mind a case Dr. Fry had under his care in which jacksonian epilepsy of segmental type began in the feet. The sensory distress was so great that the patient would pound the feet with anything she could get hold of. The jacksonian convulsions continued up the leg in segmental progression at intervals of some weeks and eventually involved the left side of the body. There was not a great deal of positive information to be gained either from the history or from the neurologic examination. She had some complete epileptic attacks. The surgeon laid down a large parietal flap, and a tumor of the falx cerebri opposite the leg center was discovered. The condition of the patient was so serious that we stopped with the expectation of going farther at a later time. The patient developed a pyelonephritis and nothing further has been done. I thought this peculiar situation of a lesion might be of interest.

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* This paper will appear in full in the *Journal of Nervous and Mental Diseases.*

Dr. L. Pierce Clark, New York: Some years ago my attention was called to the fact that many types of epilepsy in their beginning had local paralytic equivalents, and that too, in idiopathic types rather than the organic or symptomatic jacksonian epilepsy. It might be difficult to differentiate such from Dr. Spiller's cases.

Dr. William G. Spiller, Philadelphia: Dr. Clark speaks of transitory paralysis as an epileptic equivalent. I am under the impression that Flatau was among the first to describe this, and later McConnell wrote on this subject. Transitory paralysis undoubtedly occurs following a convulsive attack in epilepsy, and it may be confined to a limb, but I cannot recall a case in which this transitory paralysis, postconvulsive or as an epileptic equivalent, was confined to a segment of a limb. If it did occur, it should cause no difficulty as regards the segmental paralysis of which I have spoken.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, Oct. 16, 1919

Lewis J. Pollock, M.D., *President*

A CASE OF LUMBOSACRAL PARALYSIS. Presented by Dr. G. B. Hassin.

A colored boy, aged 18, entered Cook County Hospital about two months ago, complaining of difficulty in walking. The mother stated that when he was 4 years old, he fell on the floor with the legs widely "stretched." After this he was confined to bed for nine months, but he regained the use of his legs and for ten years did not experience much difficulty in walking, though he admits that the left leg was always smaller and weaker than the right. In the last four years the left leg has steadily grown weaker and he has often fallen because of sudden flexion of the left knee. He has had measles, pneumonia, whooping cough, gonorrhea (twice) and "five sores" on the penis (about a year ago), but at the time of the accident he was perfectly well, had no fever, headache or vomiting.

Examinaton.—Marked lumbar scoliosis to the left; atrophy and shortening of the entire left lower limb; drooping of the left side of the pelvis; a slight bend of the left knee, and eversion of the left foot were present. In walking he violently bent the body forward and to the left, swung the left leg and pelvis, the toes of the left foot striking the ground. Passive movements in the joints were possible, except in the left ankle where they were somewhat limited. On the left side flexion, abduction, adduction of the foot, flexion, extension of the toes, extension of the flexed leg, adduction and flexion of the thigh were all abolished, the only active movement possible being abduction of the thigh and flexion of the leg. The muscles implicated were all atrophied, flabby, and without fibrillary twitchings; those supplied by the obturator and posterior tibial nerves showed complete reaction of degeneration, while the muscles supplied by the anterior crural and peroneal nerve gave a normal response (only the galvanic current was available). The tendon reflexes on the left were lost, the cremasterics were very inconstant and uncertain; there were patches of anesthesia and hypesthesia scattered over the dorsum of the foot and anterolateral surface of the leg.

The roentgenographic examination revealed no bone or joint changes; the central nervous system (pupils, genito-urinary organs, mentality) was equally negative.

The clinical picture presented by this patient strongly resembled that outlined by some orthopedic surgeons (Lorenz, Bade, Peltesohn), as the result of unfortunate attempts at reduction, in small children, of congenital dislocation of the hip. Forced excessive abduction is pointed out by Bade and others as the probable cause of the lumbosacral paralysis which they think is produced by the tearing of the stretched nerves emanating from the lumbosacral plexus. The same etiologic factor was, in my opinion, the cause of the paralysis in this patient when he fell with the legs widely abducted.

FACIAL PONTILE DIPLEGIA (TRAUMATIC). Presented by Dr. G. B. Hassin.

A colored man, aged 26, had his head caught between two railroad cars. He immediately lost consciousness, which he regained, he claimed, three weeks later. About two and a half months after the injury he entered the Cook County Hospital.

Examination.—There was paralysis of both seventh nerves and of the left sixth. The face was expressionless and the lower lip hung down. The patient could not wrinkle the forehead, show the teeth, laugh, pucker the lips, close the eyes, etc. The tongue, uvula, soft palate, and the muscles that controlled chewing showed no paralysis. He complained of dryness of the tongue and mouth, had no taste for sweet, salt, bitter and sour. The seventh nerves and facial muscles showed complete reaction of degeneration. He could move the left eye in every direction except outward. Hearing was good on the left, but was somewhat impaired on the right. Douching (cold water) produced no response from the vertical canals of either ear, and there was a slow nystagmus to the left on irritation of the left horizontal canals, and past pointing to the left, 2 inches; rotation produced normal but shortened reactions from both vertical and horizontal canals (three seconds for the right and six seconds for the left ear). The roentgenographic examination revealed a fracture of the skull extending into the middle fossa, close to the base and involving the right parietotemporal region. The simultaneous involvement of the seventh and the abducens could very well be explained by a lesion of the tegmental portion of the pons where the sixth nucleus is looped by the ascending root of the facial nerve. Such a localization would also involve the longitudinal bundle and the adjacent contralateral seventh nerve which exchanges fibers with the opposite nerve. The chorda tympani changes, however, and slight hearing impairment on the right might also denote a peripheral lesion (on the right); however, this could not explain the permanent abducens paralysis on the left and the nystagmus findings.

SEX PERVERSION AND CRIME. Presented by Dr. Harry R. Hoffman.

Sex perversion is as common now as it ever has been, possibly more so. It has been estimated that there are from 75,000 to 100,000 sex perverts in Chicago. At the City House of Correction with an average of 2,000 inmates, the number of self-confessed perverts runs from fifty to 200. These persons are of such pronounced types that the attendants in the receiving room can pick them out. Nearly all of these perverts are put to work in the male laundry of the institution. The large majority are of the inverted type. Cases of exhibitionism are occasionally found, but these usually occur in debaucheés, in persons afflicted with local infirmities that explain the immodest act, or in persons afflicted with some definite mental disease, such as senile or precocious dementia. The speaker believed that many cases of prisoners sentenced for larceny and shop lifting were cases of fetishism.

The majority of the inverts in this institution show no abnormalities in physical development, but their gait, speech and tastes are feminine. They belong to the more intellectual class of prisoners. Of all the inverts now confined there, not one has failed to finish the eighth grade in school. This is remarkable considering the type of prisoners in the institution. At present there are more colored prisoners than white. These prisoners give the least trouble of any in the institution. Although the majority are recidivists, having served sentences in this or other penal institutions, they are the least troublesome. They talk freely and intimately, and they realize the depravity of their desires. They look with scorn on the other perverts, and do not care to mingle with them. The invert's love is not directed to women or children nor to other perverts, but to vigorous healthy adult males. They are intensely jealous, and it is this jealousy which often causes them to commit the crime that brings them into the penal institution.

The great majority of inverts are harmful only to themselves. Those showing criminal tendencies should early be placed in suitable institutions for mental disease. All sadists and fetishists should be handled as though they were psychoses. Those whose pervert acts are symptoms of some underlying psychosis should immediately be given institutional attention.

SOME OBSERATIONS ON DISCIPLINARY PSYCHIATRY IN THE ARMY. Presented by Dr. Herman M. Adler.

During the war, Dr. Adler had the opportunity to become familiar with the mental aspects of the problem of delinquency in the army. The army methods of dealing with delinquents are comparable to those employed in civilian life. Delinquents are confined in guardhouses pending action by court-martial, and sentences of less than six months are served in the guardhouse. Longer sentences are served in the military prisons, which are: the United States Disciplinary Barracks at Fort Leavenworth; the Atlantic Branch at Gouvernor's Island, New York, and the Pacific Branch at Alcatraz Island, San Francisco. It was estimated—on the basis of the figures for the Civil War, the Spanish-American War and the Mexican border activities—that an army of 3,000,000 men would require accommodations for 50,000 prisoners at Fort Leavenworth. Actually, the number confined in the military barracks at no time exceeded 4,000; the total number serving sentences was approximately 5,000. In time of peace, the population of the Disciplinary Barracks at Fort Leavenworth was between 1,200 and 1,700 men, corresponding to the number in the average civilian reformatory or penitentiary. Although there was an increase of population during the war, the total number was approximately 10 per cent. of the estimated number. This result is probably, at least partially, due to: (1) The enforcement of prohibition in the cantonments of the army generally; (2) the activities of the neuropsychiatric boards at the draft examinations, and (3) the distance of the actual hostilities from the home base.

The neuropsychiatric section of the Surgeon-General's Department became effective very shortly after the declaration of hostilities, and increased in efficiency continuously during the course of the war. It was not possible, therefore, for the neuropsychiatric officers to eliminate from the army all mentally abnormal men, some of whom eventually found their way to the disciplinary barracks; nevertheless, the majority of such men were eliminated at the source. As a result, delinquency was greatly reduced, as compared with any previous experience in this country. The disciplinary problems were only a small portion of the problems considered by the neuropsychiatric section and were not, therefore, developed as rapidly as other features of the work. The unexpectedly abrupt termination of the war brought these activities to an end before the effect of the various measures determined on in the Surgeon-General's Office became fully apparent. It was possible, however, to make a special study of the mental aspects of delinquency at the Disciplinary Barracks at Fort Leavenworth. A survey of prisoners, including some 3,500 cases, was made. This included: (1) complete physical and neurologic examination; (2) mental examination; (3) psychologic rating; (4) social history, and (5) study of behavior during confinement.

The prisoners examined included all the usual types found in institutions of this sort and, in addition, about 500 men confined as conscientious objectors. The result of this survey showed that from about 10 to 12 per cent. of these men were feebleminded, and that less than 5 per cent. were insane or markedly psychopathic. This low figure, as compared with figures from civilian institutions, was undoubtedly the result of elimination by draft examinations at the cantonments.

It was found that there was a correlation between intelligence rating and education. The educational medium of the entire group fell in the seventh grammar school grade. The men in the disciplinary battalion, the organization through which the prisoners might win their way back to honorable service in the army and thereby wipe out a dishonorable discharge, on the whole rated a little higher both in intelligence and education than the general average. The religious and political conscientious objectors, so-called, were the best educated of all the prisoners and had the highest intelligence rating. The prisoners convicted of sex crimes and crimes of violence had less education and rated lower in intelligence than the other groups. The men convicted of acquisitive crimes had more education than the average and had a higher intelligence rating than any other group, except the religious and political conscientious objectors. The conscientious objectors of the alien enemy and noncitizen group were very low as to intelligence and educational rating—a third of them had no schooling and one half of them had not progressed beyond the third grade.

A personality study was conducted, dividing the prisoners into three groups: (1) Those who had been in difficulty as the result of defective intelligence or judgment, or other marked mental defects; (2) those who showed no decisive defects, but whose difficulties, either in the army or previously, resulted from a marked emotional instability, and (3) those whose difficulties could be traced to marked egocentric traits.

Sixty-six per cent. of the entire group were found to be egocentric; 24 per cent. inadequate, and 6 per cent. emotionally unstable. The small number of the last group was explained on the assumption that delinquents of this type usually arouse sympathy in their fellowmen and are rarely sentenced to confinement. The inadequate group included not only the feebleminded, but the higher grades of defectives. The egocentric type expressed itself by insubordination, arrogance, an uncompromising attitude, vanity, lack of emotional generosity, and, above all, selfishness. The emotonal make-up that went with this resulted in violent or unstable behavior and was dominated by one or more of the factors mentioned above. It would appear from these studies that this type of personality was the one that most frequently caused trouble in army life, and that it was at the bottom of about three fourths of the disciplinary difficulties and delinquencies. So far as the treatment was concerned, the survey indicated that the experiences with similar personality disturbances observed in the hospitals for mental diseases were applicable. The inadequate group required suggestion from a stronger and more intelligent personality. The emotionally unstable group required care during their emotional upsets, and reassurance and discipline of habit at other times. The egocentric group were best managed by an impersonal and logical attitude on the part of the authorities. Any attempt to change the type of personality, either by logical argument, or by coercion with force or otherwise, led to an increase in the egocentric manifestations. The sane method of persuasion and of impersonal attitude toward the inviolability of individuality in the end secured the prisoners' concurrence, if not their cooperation.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, Nov. 20, 1919

George A. Waterman, M.D., *President*

THERAPEUTIC PROCEDURES FOR THE RELIEF OF DECREASED INHIBITION. Presented by Dr. John Bryant.

In animals, and in man, life is difficult or almost impossible without reasonably normal development and action of the central inhibitory mechanism. The world is full of patients, mostly chronic, in whom inhibition is decreased. One obvious, ultimate result of this decreased inhibition is chronic exhaustion. In order to relieve chronic exhaustion, its cause must be attacked. In diet and exercise, properly utilized, we have two valuable and apparently too little appreciated methods of attack on the problem of decreased inhibition. The first essential in the matter of diet is the temporarily complete elimination of eggs, meat and fish, and the restriction of milk to a few ounces daily. This strict diet is maintained for from two weeks to a month. At the close of this period, there is a gradual resumption of general diet, but on an intermittent basis. The second week there may be two days of unrestricted diet, and in the third progressive week the patient may arrive at unrestricted diet every other day. It is unlikely, however, that such a patient can ever profitably take unrestricted diet on continuous days for any great length of time without suffering harm from excessive chemical stimulation. The employment of physical exercise has for its immediate object the sharpening of muscle sense perception. Such exercise is carried out at the very slow rate of about ten seconds per linear foot of distance. One simple exercise for each main group has been developed, and in the army a routine was employed which took for its full completion about one hour of time. Parallel with the increasing power of physical control comes a noticeable increase in mental poise and the evaporation of mild phobias which have as their basis the inabilty to handle properly the locomotor mechanism. The two most usual phobias observed have been the fear of high places and the fear of crossing streets when there is city traffic.

DISCUSSION

Dr. Morton Prince: I wonder if Dr. Bryant would state more clearly and in detail the class of cases to which he refers.

Dr. John Bryant: The patients that I have been working with are mostly the asthenics and the enteroptotics. They are almost always in a state of chronic exhaustion. They come to me because they have some intestinal trouble, but as I work with them I find that I must consider the general physical development and eliminate some of the mental abnormalities before I can produce results. I saw this afternoon a working man who, when he came to me, could not get through his day's work. Now, after two months, he is hard at work and goes home feeling fresh instead of exhausted, and he sleeps soundly instead of staying up all night complaining of his gastro-intestinal tract.

Dr. E. W. Taylor: One of the chief values of this communication, it seems to me, is what we have been seeing recently in so many branches of medicine—closer relationship between the neurologic side of disease and certain definite physical phenomena which ordinarily have been included in the general category of internal medicine. On every hand, we are observing this integration, as it were, in these branches of medicine, particularly in relation to the nervous system. No doubt the war has had much to do with bringing this out.

TRANSMISSION OF BREATHING THROUGH THE HEAD. Presented by Dr. A. Myerson.

In various parts of the head, and especially in persons without much hair, breathing sounds can be easily heard with a stethoscope applied in the usual manner. The points at which they can be heard best are: First, over the antrum, where they are loud; second, over the temples where they are less clear in some individuals, but where in many cases they are quite distinct; third, on the vertex, where in bald-headed men they are as distinct as over the temples; fourth, on each side of the occiput; and fifth, just below the occiput in the upper cervical region, near the spinal column where they are very distinct. The sounds heard are blowing in character, with prolonged expiration. It is sometimes necessary to ask the subject to breathe deeply. The sounds are unquestionably transmitted directly through the skull, and very likely come from the upper nasal, pharyngeal and laryngeal parts of the respiratory tract. This is reported as a purely physiologic phenomenon constantly present whenever conditions make it possible to get close to the skull. No pathologic relationship has as yet been investigated.

NEUROLOGIC AND PSYCHIATRIC WORK AT SAVENAY. Presented by Dr. H. M. Swift.

The hospital center of the American Expeditionary Forces at Savenay comprised a number of base hospital units, one of which was Base Hospital No. 214, used exclusively for the care and evacuation of patients whose cases were previously diagnosed as nervous or mental. The types most frequently seen were those of mental deficiency, various psychoses, psychoneuroses, epilepsy and constitutional psychopathic states. There were also admitted many patients with organic nervous conditions, of which perhaps the most noteworthy were the residuals of encephalitis lethargica.

The mental defectives might be roughly divided into the physically strong and the physically weak. Previous to the armistice, mental defectives of good physical condition and without conduct abnormality were usually reclassified and recommended for labor organizations. Frequently, mental deficiency was found to be associated with congenital physical deficiency manifesting itself by generally poor bodily structure, often with narrow chest, stooping shoulders and flabby abdominal wall (the type designated by some as the carnivorous type). For these persons with associated congenital mental and physical deficiency the term constitutional deficiency would seem more descriptive than mental deficiency and would not imply so great a stigma. In this group, evidence of a generally poor nervous system was shown by the small resistance to stress and fatigue with resulting neurasthenic states.

The psychoses did not differ strikingly from the psychoses of civil life. Interesting were the cases of soldiers with clinical pictures resembling that of dementia praecox who subsequently made apparent recoveries. Of manic depressive insanity, maniacal states were relatively more common than among a corresponding male population in civil hospitals for the insane.

The psychoneuroses comprised the war neuroses, or those arising in connection with shell explosion, and neuroses occurring independent of so definite a causal factor. The latter did not differ essentially from the neuroses of civil life. In both groups, the constitutional element was frequently evident.

Epilepsy: Of especial interest were cases of epilepsy developing in the service with a history of no attacks previous to enlistment. It is suggested that in addition to the ordinary type of epilepsy manifesting itself in the form of convulsions, etc., there may also exist persons with a latent epileptic tendency which under ordinary conditions may never come to the surface, but which becomes active under especially unfavorable influences, such as may be incident to army life.

The term constitutional psychopathic state was rather loosely employed to designate various character abnormalities. In this term were included the cases of many men who by reason of native peculiarity or defect showed themselves inadaptable to army life. Alcoholics and many delinquents were also classified under this heading.

Encephalitis Lethargica: At Base Hospital No. 214 only residuals were seen, chiefly cases of cranial nerve palsies and other cases of a peculiar condition resembling paralysis agitans with stiffness and with mask-like facies. The cranial nerve palsies were undoubtedly nuclear. The oculomotor and the facial nerves were most frequently affected. The frequency of nuclear lesions suggests that the inflammatory process has a tendency to damage more particularly gray matter. The state that resembled paralysis agitans might be explained perhaps on the basis of damage to some hypothetical nucleus that normally has a regulatory influence on the motor system, the lesion leaving as a resultant the peculiar symptoms noted.

Several cases of flaccid paralysis of the legs following infectious disease were seen which were diagnosed as peripheral neuritis, but which might have been due to an anterior horn lesion. They differed from the ordinary peripheral neuritis of so severe a grade in the rapidity of improvement; in this respect they resembled some of the cranial nerve palsies.

May it not be that the inflammatory process of encephalitis lethargica is really of the nature of a polioencephalomyelitis and that the symptoms vary according to the region of the central nervous system most seriously affected? In many of the cases a history of a lethargic state could be obtained. This seemed sometimes to have followed influenza. In other cases the history was indefinite. In one case of the paralysis agitans type, although the stiffness seemed to have been of sudden onset, the patient maintained that otherwise he had not been ill. Yet the clinical picture was almost precisely the same as that in other cases with a definite history of an acute febrile state.

NEUROPSYCHIATRIC EXPERIENCES AT VICHY AND SAVENAY.

Presented by Dr. Donald J. MacPherson.

During the months of October, November and December, 1918, 124 brains and 119 spinal cords were examined and preserved at the Neuropathological Department of the Central Laboratory at Vichy. In twenty-two cases gross lesions were observed. There were eleven cases of meningitis, of which seven were secondary to gunshot wounds of the head or spinal column. In three cases, injury of the lumbar or sacral portion of the spinal canal was followed by a diffuse meningitis involving the cord, and base and vertex of the brain. Five cases of pneumonia were complicated by meningeal hemorrhage. In one case presenting the clinical picture of a transverse myelitis with paraplegia, following penetration of the back one-half inch to the left of the vertebral column by a small piece of shrapnel, the vertebrae were found uninjured and the dura intact. One and a half inches of the cord in this region appeared as a soft, flattened mass. There was indirect traumatic softening. Other pathologic findings included cases with tetanus, syringomyelia, pituitary tumor and one with a small cholesteatoma.

An analysis of 1,760 cases classified as mental or neurologic at the Savenay "triage" to March 1, 1919, showed 1,436 injuries to nerves of the extremities. Of these, 609 were on the left side of the body, 544 on the right side, and 283 were not classified as to location. Graphic charts showed the relative greater frequency of injuries to nerves of the upper extremities. There were eighty cases with injuries involving the central nervous system, twenty-nine injuries to cranial nerves, and fifty-six miscellaneous affections including syphilis, epilepsy, multiple sclerosis, brain tumor, neuritis, etc. Only twenty-nine functional cases and one psychosis were reported. One hundred and twenty cases were unclassified. These were largely cases in which complete examination was difficult because of splints.

Dr. John Bryant: I was asked whether I would describe the kind of patient that I was dealing with in this paper. I think the type has been well described by Dr. Swift. It is the same type of patient as that of which he speaks—the congenitally deficient patient—only he is referring to those in whom the mental element is more pronounced than the physical element. The similarity of the two types of cases is evidenced by the fact that Dr. Swift described them by the word "carnivorous," a term for which I am primarily responsible.

Dr. George A. Waterman: This question of discipline was one of the most difficult problems with which we had to deal at Parker Hill. We were often asked to see a man reported because, for example, he had given the sergeant a knock-out blow. Those things were fairly simply dealt with when they occurred near the front and when the army was in operation, but when the soldiers reached us they were simply waiting to be dismissed and discipline had become very lax. They were more or less filled with the idea that they were heroes, and the attitude of the commanding officer was that strict discipline was sure to bring newspaper notoriety. The men wanted to be dismissed and it was very difficult to send them to hospitals for neuroses. We had men from the tenderloin, for instance, who had never had any discipline and who were distinctly of a psychopathic constitution; such men were very difficult to manage.

Dr. J. W. Courtney: I should like to ask Dr. Swift, first, whether in his rather large group of cases of encephalitis lethargica there were fundus examinations and if so, what those examinations revealed; and, second, as to what the exact syndrome was in those cases which were afterward recorded as "psychoses, unclassified," but which were referred to him under what I consider the most ill-chosen and unfortunate of all psychiatric designations—dementia praecox.

Dr. H. M. Swift: In the encephalitis lethargic cases, the fundus when examined was found normal. The symptoms in the cases resembling dementia praecox varied greatly and many times were fairly well cleared up when the patients reached us. We sometimes had a history of a catatonic condition. Other patients were more of a paranoid type, that is, they had delusions, not very well systematized, which very frequently improved. For that reason, we were always reluctant about making the diagnosis of dementia praecox because that rather implied the disease had existed previous to enlistment and we thought that those patients in whom the condition had developed while they were in the service were entitled to compensation as for an injury received in line of duty.

Dr. Morton Prince: I should like to ask a question; but before I ask it I should like, apropos of dementia praecox, to mention a case. I think it was one of the worst cases I have ever seen. The patient was sent out to Waverley, and after being studied a long time the diagnosis of dementia praecox was made. It was a terrible case, so bad that the family was told that one could only hope that complete dementia would come soon and so end the mental distress. Recently, while overseas, I had a letter from the young lady saying that she is perfectly well. (Dr. Waterman, who also was concerned in the case, confirms this report.) Hence a patient with a most severe case of dementia praecox may recover.

The question I was going to ask is this: At the last meeting of this Society that I attended before going abroad, the subject of examinations to weed out the feebleminded from the drafted men was discussed. I recall that the members of the Society were explaining with some enthusiasm the results of their methods for the detection of cases of mental inferiority, etc. I also remember that Dr. Fernald said that about twenty-seven of his patients had been accepted by the army. Dr. Knapp then asked "if he had hung out a service flag from his institution?" How was it these morons of whom Dr. Swift speaks got into the army? Were the bars let down or were they not detected in spite of the mental tests when they passed their examinations in various parts of the country? How was it they got in, and when they were in did they do good work? You said some were sent to the army labor organizations. The psychiatrists on the examining boards were reporting their wonderful results, and the neurologists were reporting their results, and here we have these large numbers of defectives in the army.

Dr. W. H. Prescott: In this connection I may say that we were asked to see a patient at the psychopathic hospital whom we found to be a man that had escaped from the Worcester Hospital three months before, and that had been admitted to the army within a month from the time he had escaped. He was a most marked example of dementia, and did not seem to be able to tell where he had been or what he was doing. He was immediately sent back to Worcester. How he got into the army I have never been able to understand because his face showed what he was.

Dr. A. E. Brownrigg: Apparently all these cases of mental deficiency and other abnormalities were not detected on the first examination by army physicians, and yet I think those who have seen a great many cases of mental deficiency in the army cannot help looking with admiration at the results that have been achieved by the neuropsychiatric units. I was stationed at Fort Porter. Here were received practically all the patients suffering from mental disturbances that came to this country, and I presume that individually I saw more of these returned patients than any man in this country. In considering these men, a large number, as Dr. Swift has intimated, were found to be mental defectives. I have no statistics, but I have a clear memory that the majority of these patients came from the South, Louisiana and Mississippi, and also a large group came from Minnesota and Wisconsin. I gathered from correspondence in trying to place them back in their homes and from their service records that most of these patients came into the army before the neuropsychiatric units were operating. We investigated these cases time and time again and found that these men were sent overseas in the early months of the war. We know that Dr. Pearce Bailey and the members of his department were not actively concerned in examining these units until late in 1917 or early in 1918. I think, therefore, that of the troops that came through in 1918 that received the neuropsychiatric examination, we did not receive any of the low grade imbeciles, and it was only in those early enlistments that we occasionally saw them. I remember one patient in particular. I sent him home with an attendant because he did not know enough to travel alone. He said papa had always driven him to town. He graded six years and had never been allowed away from home. He was 37 years old, and anybody would know that he was a born fool. He was not, of course, a good soldier, and he spent his time in hospitals.

The psychopathic patients were called "incorrigibles;" they were not adaptable to army life and it was very hard to have them discharged honorably.

The patients with dementia praecox had all the cardinal symptoms of dementia praecox under the old classification of Kraepelin. They were sometimes brought to us on stretchers and with special guards. Some were paranoid and some were of the simple dementia form. But the remarkable fact was that after a stay of three or four months, a large proportion of them became well enough to be returned home, sometimes without an attendant, and they made a very good social recovery. Many years ago I read a paper before this Society in which I suggested the improvement and cure of dementia praecox. I never thought that the dementia in dementia praecox meant that a certain patient was always absolutely demented, but that he had dementia praecox in much the same real way that one might be without severe typhoid symptoms, but nevertheless have a true attack. I think that this experience in the army made me believe that whether considered to be a psychogenic disease wholly or one that is on a defective basis, the patients that lose control of their minds are curable and recover if treated early. I think this experience in the army has proved the truth of such a general statement.

Dr. H. M. Swift: In answer to Dr. Prince, I should like to say that although we had a great number of mental cases, both defective and psychotic, yet relative to the total number of men who went over I think the number was probably rather small.

Book Reviews

DIE HYSTERIE ODER SOGENANNTE PSYCHOGENE NEUROSE: Eine psychopathologische Studie auf dem Grenzgebiete des Nerven-und Seelenlebens. By Oscar Lessing, Dr. phil. et med., Arzt in Berlin. Berlin, 1920, S. Karger, p. 45.

This is an educational essay for the laity presenting generally accepted conceptions of the nature and symptoms of hysteria. Because of the enormous increase in the number of cases of hysteria caused by the great war, the author thinks that the people at large should know more of this nervous disorder. The diction is diffuse, but readable, and the matter is sound.

TRAUMA UND NERVENSYSTEM: Mit gleichmässiger Berucksichtigung der Kriegserfahrungen der Ententeländer und der Mittelmächte. By Dr. Th. van Schelven, Neurolge der Niederlandischen Sanitätsmissionen. Berlin, 1919. Verlag Von S. Karker, p. 303.

This work is a systematic presentation of our knowledge of traumatic injury of the brain, cord and peripheral nerves with a brief (twenty-four pages) consideration of traumatic neuroses. The practical basis of the book is the author's war experience. As a neutral he had the advantage of observation on both sides of the line, and he avows an effort to present the sum of international knowledge of trauma of the nervous system acquired during the war. As a summary the book is valuable but it lacks the punch that is given by an author who is himself master of the subject. That the literature has been exhaustively consulted is evident, but too often the name of an author is given without the bibliographic reference. For instance, on page 3 authors are referred to twenty-two times with only six indications of the source in the literature. Many words are wasted in statements of the obvious. One scarcely needs to be told that "long continued compression, which need not be very intense, may cause nerve paralysis," and that "gunshot injuries of the skull belong to the most dangerous wounds of war and peace." All of this gives the work a pedantic air and the reader the impression that the author either is not quite sure of himself or is a beginner in book writing.

Although war work apparently is responsible for the book's having been written, it is in no sense to be regarded as a work on war pathology and practice, but it is an attempt to cover peace trauma quite as completely. The entire subject is treated in the most methodical way. The section on peripheral nerves (107 pages) takes up in succession pathogenesis, pathologic anatomy, clinical manifestations, diagnosis, therapy and prognosis. Under diagnosis there are nine subheads. The same painstaking method is pursued throughout so that the student is sure to find every phase of the subject considered. The work contains a great amount of information and summarizes a vast deal of knowledge. What is lacking is discriminating emphasis. The book is an honest, solid product of considerable value, but the tyro will have difficulty in separating the essential from the nonessential and the ordinary from the exceptional. The experienced neurologist or surgeon will find much that is superfluous.

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MORBI NEURALES

AN ATTEMPT TO APPLY A KEY PRINCIPLE TO THE DIFFERENTIATION OF THE MAJOR GROUPS *

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This paper is intended to be more modest than even its subtitle "attempt" denotes. The writers feel confident that a key principle, roughly reminding one of the principle of Gray's botany for the diagnosis of plants, is applicable to the diagnosis of nervous diseases. Central in the application of such a key principle is the idea of exclusion of diseases in diagnostic consideration by means of following a certain order. But granting the theoretical virtues of a key principle and of the method of orderly diagnosis by exclusion, we must remain uncertain whether the order adopted is the *one best way,* in the sense of the efficiency engineer who devised that term.

The attempt to apply a key principle to the differentiation of the major groups of nervous diseases was based on the comparative success of the application of an identical principle to the differentiation of the major groups of mental diseases. Since the publication of a key to the practical grouping of mental diseases, presented to the American Neurological Association in 1917, the principle of diagnosis per exclusionem in ordine has been presented to the Association of American Physicians in 1918, and in briefer form in a second paper on certain applications of the pragmatic method to psychiatry (1918). The idea of a possible extension of this principle of orderly exclusion to the problems of general medicine has met with the approval of Dr. Richard C. Cabot, who, at the June, 1919, meeting of the Association of American Physicians, displayed a tentative list (without definitive sequence) of the major groups of diseases in general. This list, as the sequel will show, may have some value for clinical neurology, and may even prove superior to the key to mental diseases formerly laid down.

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* Read by title at the annual meeting of the American Neurological Association, held at Atlantic City, N. J., June 16-18, 1919.

DIAGNOSIS BY EXCLUSION

Perhaps it is superfluous to argue here for the virtues of the key principle in the form of diagnosis per exclusionem in ordine. In a previous paper considerable argument has been devoted to the habitual underestimation of the values of the method of diagnosis by exclusion, of which DaCosta and his followers were guilty in the sixties and seventies of the last century. The method of diagnosis by exclusion was to be resorted to only in extremis, according to these authors, and especially according to the book on diagnosis by Da Costa, published in 1864, which was thought by some to have marked an epoch in the progress of internal medicine. The so-called indirect diagnosis or diagnosis by exclusion was, according to DaCosta, "not on ordinary occasions much employed, nor indeed," DaCosta continues, "is it to be recommended." DaCosta thought that to prove what a thing is by proving all that it is not is a very tedious process. He went on to say that it was "difficult to think of all the possibilities" and remarked that "pathology was in an imperfect state." But its imperfections seem to us to interfere with all other methods of diagnosis as much as with diagnosis by exclusion. As for the difficulty of thinking of all the possibilities, the device of placing these possibilities in manageable groups (after the manner of the diagnostic groups of animals, plants and minerals, that is, after the manner of Linnaeus), DaCosta and his colleagues in the single-volume textbooks of medicine did not conceive of such a process. Apparently, DaCosta felt that in the process of diagnosis by exclusion one would need to exclude theoretically every other disease than the one on which one would finally fix. As for the tediousness of this process of diagnosis, no doubt accurate diagnosis in difficult cases is and must remain extraordinarily tedious, but remains none the less interesting and important.

But diagnosis by exclusion is not diagnosis by exclusion in order. It is one thing to secure a list; it is another thing to make a sequence of the elements in the list. An aggregate does not forthwith throw itself into order, and especially not into that arithmetical arrangement called a "well-ordered" aggregate.

Now as a matter of fact, it seems to us that most workers in difficult cases do actually employ something like what has been described as diagnosis per exclusionem in ordine. In correspondence with some specialists, affirmative replies have been given to questions on this line. Ordinarily, diagnosticians are prouder, perhaps, of making a diagnosis of smallpox by the sense of smell at 100 yards (more or less) than to make a careful, logical diagnosis by exclusion in order. Yet the most expert diagnosticians are reduced to using diagnosis per exclusionem from time to time. Even DaCosta admitted that the method had to be used on occasion. We hold that a method which has to be resorted to in difficult cases ought to be rationalized so that tyros and inexpert workers may the earlier learn to make correct diagnoses. We hold that the field of mental and nervous diseases is a good field in which to begin, because the majority of cases in these fields are difficult of diagnosis. Especially in the field of mental diseases is it true that there is practically no single pathognomonic symptom in the whole branch. Starting with a slant toward some disease in which a given symptom is characteristic, the tyro can prove from the very best textbook that almost any mental case he has in hand is or might well be a case of the disease he starts to prove it to be. The tyro is only caught up short when the laboratory comes to the rescue with pathognomonic signs. But the tyro with a slant rushes in where the laboratory expert fears to tread. On this account, as there are practically no indicator symptoms in the field of mental disease, the method of diagnosis per exclusionem in ordine was found to have especial value for the mental diseases.

We shall not here rehearse the situation in the mental disease groups. We may persist in pointing out, however, that, if eleven groups of mental disease have been laid down as of pragmatic orderly value in diagnosis, there is no reason why some other worker may not prove that there are twelve or ten instead of eleven groups. Thus one acute observer believes that there should be added to the eleven-group system, as presented, a twelfth group of paranoic cases. Another acute observer suggests that there are really no senile diseases, that properly obtain the name, at least among the mental diseases, and that consequently the senescent-senile group should be deleted from the sequence and its diseases distributed otherwise; for example, among the arteriosclerotic brain diseases, the manic depressive group, etc. Let us insist, too, that not only may the groups be accordeonized, that is, interpolated and extrapolated without destroying the key principle involved, but the order of the groups may likewise be changed, possibly to advantage. Some considerations on this point have been given in the paper above mentioned on applications of the pragmatic method to psychiatry.

CLASSIFICATION OF DISEASES

For those who are willing to consider a rationalization of the method of diagnosis by exclusion in order (a method which we are convinced that every one dealing with complex cases must in some form employ) there is another bogey of greater difficulty. Pathology, as DaCosta might still remark, is in an imperfect state! Why classify diseases in any definite manner until we know more about the etiology and genesis of diseases? The answer to this objection is that in practical life some account of stock must continually be taken. Next year the very basis of some well-recognized disease of today may be entirely overthrown; even today we may recognize that the etiology or genetic basis of that particular disease is very shaky or rickety. Nevertheless, it remains our duty to make diagnoses on the best basis available; otherwise there will be no common ground of discussion with colleagues and no common ground for practical measures of treatment. Accordingly, the imperfections of present day pathology should not deter us from practical classifications. We must cheerfully admit that these practical classifications may be changed from decade to decade. The pessimist or the worker weary in well-doing, may regret the long string of obsolete and obsolescent classifications that the world provides, but there is no genuine ground for such regret where the classifications have been made in good faith by careful and logical thinkers. Some authors appear to think that there will some day be a single classification of diseases, and every day we see classifications put forward by eager workers which they seem fondly to wish shall replace all other classifications. A glance into logical works on the topic of classification, even into the ancient work of John Stuart Mill called a System of Logic, would reveal to these workers the infinitude of classifications of the very same objects. A classification by causes may totally differ from a classification by genesis, and both may differ from a classification for the purposes of diagnosis, and in the end a classification by methods of treatment may be found to cut quite across all other classifications. Then, too, it is probably worth while to devote a sentence to the universal occurrence of the editorial imbecile (or shall we say idiot?) who makes fun of what he calls the classifying mania, apparently not being aware that almost the whole development of science and art depends on this so-called mania.

The etiologist may have in mind some particular factors which he would like to elevate into the so-called causes of disease. At the other end of the line some hospital administrator will be found who would like to classify all cases according to the amount of food, nursing and open plumbing which they may require. In between these two extremes may be found the practical diagnostician who has troubles of his own. The present effort deals with the troubles of the diagnostician rather than with the troubles of the etiologist or of the hospital administrator.

With more particular respect to the nervous diseases themselves is it not possible that the nervous diseases will very shortly fall into a common group with the mental diseases? Is there any point in producing an order of nervous diseases independently of an order of mental diseases? Probably the new art of neuropsychiatry is on its way, and neuropsychiatrists with equally good training on both sides will become available. However, we believe that it is the pragmatic situation at the present time that psychiatrists and clinical neurologists are two separate and different kinds of specialists. No doubt the fundamental basis of both arts lies in the science of neuropathology, taken in its widest sense of including both structural and functional concepts—but that wide and deep science is still (to borrow a leaf out of DaCosta's vocabulary) in an imperfect state. The question is a practical one. It is something like the question whether we ought to separate the specialties of dermatology and syphilis or the specialties of surgery and gynecology. Now one opinion, now another, will prevail, and there will be a progress toward a final decision.

Classification of Nervous Diseases.—As we approach the topic of the nervous diseases taken in their general sense, we find that the distinction between "organic" and "functional" nervous diseases is the most fundamental one in many systematic works and in perhaps the majority of nonspecialistic medical minds. Is a case organic or is it functional? — this is about as far as the general practitioner likes to wander in the field of clinical neurology. He has an idea that by means of the "reflexes" the neurologist more or less effectively arrives at a diagnosis of organic nervous disease and is often somewhat astonished that the neurologist cannot, by doubling or trebling the number of "reflexes" taken, somehow arrive at a diagnosis doubly or trebly accurate. In our view, this no doubt perfectly true distinction between organic and functional diseases is of little diagnostic value in the individual case, and this is aside from any doubts we may have as to the definition of the term "functional."

We examined the nervous diseases in sundry systematic works and seemed to be able to put these diseases into five groups as follows:

Tentative sequence of major groups of nervous diseases due to

1. Infection.
2. Historrhexis.
3. Neuronatrophy.
4. Imbalance.
5. Special.

Application of Classification in Diagnosis by Exclusion.—Another word as to its application by the tyro or by the general practitioner. Let us suppose the general practitioner confronted by a paralysis. Suppose in the course of his thinking he lights on the hypothesis that it is due to "neuritis." He may then consider or look up in the books all the types of neuritis which he can find and look for signs or symptoms appropriate to one or the other of these groups. Now, of course, the diagnosis of neuritis may in itself be erroneous to start with, and the inquiry as initiated may become useless. According to our ideas, it would be much more to the advantage of the patient if the diagnostician proceed somewhat as follows: Here we have a case of paralysis of a limb! In the first place, is this paralysis at all related to infection? Are there any signs of (1) infection whether in temperature, in blood, or otherwise, in this patient? What tests should be employed to secure evidence on the point? In the second place, if the case shows no sign of infection let us turn to the question of injury or other lesion of whatever sort which may have affected the tissues from without. Even if we cannot absolutely exclude infection as a matter of fact, let us for the purposes of diagnosis exclude it as a matter of argument until the rest of our tests have become available. Excluding infection, is there any evidence in the situation of a condition to which we above gave the name (2) historrhexis? Historrhexis might include the severing of a nerve or the nerve tissues by a knife or a bullet. It would include the blocking of nerves and the by-effects of historrhexis. For diagnostic purposes it would include the effects of an apoplexy not only as directly due to the destruction of brain tissue, but also to the pressure on collateral tissues. Not only trauma and arteriosclerosis, but also tumors, would be included in this concept of historrhexis, from which for our purposes the orderly exclusion would have already excluded infection. The historrhexis inquiry sums up to an inquiry for trauma or other tissue-injuring lesions of noninfectious nature and of a focal character.

Thirdly, the diagnostician, having got rid of the hypothesis of (1) infection (including, of course, neurosyphilis) and of trauma and other forms of (2) historrhexis, would arrive, according to our scheme, to the group which we have termed (3) the neuronatrophies. Our first thought was to name these the "scleroses," since the group contains the various classic scleroses, like lateral sclerosis, multiple sclerosis, etc. But, on the whole, the primary fact appears to be that of neuronatrophy, a condition not of the general nature of historrhexis, but of a much finer and more differentiated type. With the infections and the common historrhexes out of the diagnostic way, the tyro would now come to the neuronatrophies, that is, to questions of combined system disease, syringomyelia, multiple sclerosis, etc. (The fact that, e. g., syringomyelia often results in historrhexis with secondary degeneration, should not bother the diagnostician a whit. He has already considered the historrhexes and eliminated perhaps a transverse myelitis from the field of diagnosis. He does not arrive primarily at the question of syringomyelia, but only after having eliminated the effects of historrhexis [pressure myelitis] and infections [Pott's disease] which should be discriminated from syringomyelia.) (Again, some one might prove that multiple sclerosis is infectious. But the infectious group of nervous diseases, excluding some peripheral forms, is essentially a group of diseases in which pleocytosis of the spinal fluid is characteristic diagnostically. Multiple sclerosis appears to be in general outside this group. For the present pragmatic purposes multiple sclerosis should evidently fall among the classic neuronatrophies rather than among the historrhexes or infections of the nervous system.)

Fourthly, if the diagnostician has been able to eliminate (1) infections, (2) rhexes of tissues, and (3) differential atrophies of neuron system, he will arrive at a somewhat nondescript group which we above termed (4) imbalance, under which the drug-neuritis cases, sundry metabolic disorders, the endocrinopathies and sympathetic system disorders fall. A great many of these conditions are conditions of what we might term tissue-imbalance. Imbalance is particularly well shown in the endocrine and in the various sympathetic disorders, and may also be shown in the metabolic group in some sense. Whether multiple neuritis should not sometimes be grouped among the infections and sometimes among metabolic conditions is a question which we do not care to take up. Our purpose is not to add to theories concerning causation or genesis, but merely to take the diseases as they stand in the systematic works and throw them into an effective diagnostic order for the purposes of orderly exclusion.

Fifthly, over and above the (1) infections, (2) historrhexes, (3) neuronatrophies and (4) imbalance groups we have (5) a special or miscellaneous group in the field of nervous diseases, including such conditions as the various aglias, migraines, tics, spasms and vertigo, to say nothing of myopathies, osteopathies, various tissue atrophies, unexplained hydrocephalus, etc. With respect to this last group, the modesty of the clinical neurologists must forbid their complaining about how little they know. The aim of the present classification of nervous diseases is not to display the ego of clinical neurologists, but to find a method for arriving at diagnoses in a more logical manner.

Comments on This System.—We conceive that a system like that of classification into these five groups will serve to clarify many problems in the tyro's mind. The expert may not need it. We suspect that the expert is already using some such method, perhaps one private to himself which he has never thought of betraying to ordinary man. In conversation with various diagnosticians we find that they habitually use such systematic works as Oppenheim in a manner peculiar to themselves. Diseases in a book like Oppenheim's are worked over precisely as if they were in no important order whatever. We do not deny that Oppenheim's excellent work arranges the diseases in an order on a certain basis. We deny that the order adopted has much to do with practical diagnosis. Practical diagnosis in clinical neurology appears to have been chiefly a topical diagnosis devised to find, in the brain or spinal cord, the seat of lesions chiefly of the group we have above termed historrhexes. To adopt a more general medical attitude such as that defined in these five groups is a plan not found in textbooks of clinical neurology. We suspect, however, that the more mature the clinical neurologists become, the more they arrive at some such practical method for diagnosis by exclusion in order.

We have no idea of insisting that the order we here present is a final order, that it is an order which forbids interpolation or extrapolation, or that it is an order that cannot be changed to advantage. We would like to have the topic considered carefully by those who are concerned, and we are of the opinion that some uniformity of the scheme of elimination for practical diagnostic purposes for the various nervous diseases will greatly benefit the treatment of patients. If, by the naked eye, one has somehow guessed that a patient has a nervous disease, perhaps by means of the low power of the logical microscope, he will come to one of the five groups above mentioned as the logical site of the disease in question. Suppose now that he turns on the somewhat higher powers of the logical microscope. The five groups are subdivided somewhat as follows:

Subdivision of Five Groups of Nervous Diseases

1. Infections
    - Treponema
    - Bacteria
    - Parasites
    - Unknown infections

2. Historrhexes, focal destruction (non-infectious)
    - Trauma
    - Vascular
    - Heightened intracranial pressure

3. Neuronatrophies, scleroses ("classical degenerations")
    - Systemic scleroses
    - Diffuse scleroses
    - Hereditary scleroses

4. Imbalance exo(neuro)genous
    - Drug
    - Metabolic
    - Endocrine
    - Sympathetic

5. Miscellaneous
    - Algias, migraine, vertigo
    - Tics, spasms
    - Myopathy
    - Osteoarthropathy
    - Tissue atrophies
    - Hydrocephalus
    - *Et al.*

SYNOPSIS

1. INFECTION

(Diagnosis by signs of infection, pleocytosis of liquor, etc.)

Treponema	*Bacteria*	*Unknown*
Tabes	Epidemic meningitis	Poliomyelitis
Pachymeningitis cervicales hypertrophica	Tuberculous meningitis	Hydrophobia
	Streptococcus meningitis	Landry's disease
General paresis	Neuritis	Herpes zoster
	Tetanus	Chorea minor
	Myelitis	
	Encephalomyelitis	
	Abscess	

2. HISTORRHEXIS (FOCAL DESTRUCTION)

(Diagnosis by reflex-signs, heightened intracranial pressure, etc.)

Trauma	*Vascular*	*Tumor*
Caisson	Little's disease	Recklinghausen's disease
	Hematomyelia	
	Anemia	
	Hyperemia	
	Hemorrhage	
	Softening	
	Hemorrhagic encephalitis	
	Polioencephalitis, acute hemorrhagic, superior	
	Sinus thrombosis	
	Aneurysm	
	Pseudobulbar palsy?	

3. NEURONATROPHY

(Diagnosis by characteristic picture and history)

Systemic Sclerosis	*Diffuse Sclerosis*	*Hereditary Sclerosis*
Lateral sclerosis	Multiple sclerosis	Friedreich's ataxia
Combined system	Pseudosclerosis?	Amaurotic idiocy
Amyotrophic lateral sclerosis	Gliosis spinalis	Familial infantile paralysis
Primary muscular atrophy	Syringomyelia	Primary muscular atrophy, familial form (bulbar form)
Senile paraplegia	Paralysis agitans	
		Huntington's chorea

4. IMBALANCE

(Diagnosis by characteristic picture)

Drug *	*Metabolic Disorder*	*Endocrinopathy*	*Sympathetic*
Neuritis	Neuritis	Spasmophilia	Acroparesthesia
Multiple neuritis	Diabetic, senile, etc.	Tetany	Angioneurotic edema
			Myxedema intermittent
		Exophthalmic goiter	Hydrops articulorum
		Cretinism	Raynaud's disease
		Infantilism	Erythromelalgia
		Acromegaly	Scleroderma

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* It is possible that these cases should be placed under historrhexis, although many of the phenomena are reversible.

5. Miscellaneous

Algeas

Migraine

Vertigo

Tics—Spasms
Occupation-neuroses
Tic convulsive

Myopathy
Primary myopathy
Dystrophy, muscular progressive
Thomsen's disease
Myasthenia gravis
Paramyoclonus

Osteoarthropathy
Vertebral disease
Luxation—fracture

Caries
Tremors
Syphilitic
Arthritis deformans
Osteomalaceous paralysis

Tissue Atrophies
Hemiatrophy facialis
Hemihypertrophy facialis

Hydrocephalus
Meningitis serosa

Undefined
Caisson
Anomalies
Reflex paralysis
Exhaustion paralysis
Periodic paralysis

SUMMARY

The method of diagnosis by orderly exclusion, already proposed for use in the diagnosis of mental diseases, is probably of equal value in the field of nervous diseases. The writers have endeavored to gather the main types of nervous disease into a comparatively small number of groups for successive consideration by the tyro, or even by the expert, in diagnostic elimination. Experts may prefer a different order from the one proposed; but it is unlikely that any neurologist fails to use, consciously or unconsciously, some form of orderly diagnosis.

Yet the student is quite likely to be taught that the best procedure is to pick out some striking symptom in a case under consideration, and to follow that symptom back to textbook models for a suggestion as to the entity involved. He then endeavors to match the data of the case in hand with the possibilities laid down in the textbook.

We think that it is much more desirable to take the general situation in the body at large into account, and to include, if possible, first, the hypothesis of an infectious origin for the symptoms. Secondly, we try to exclude, if possible, the effects of coarse and otherwise destructive lesions of the nervous system (historrhexes) namely, in general such conditions as show no signs of infection, but exhibit reflex disorders and signs of heightened intracranial pressure and the like, suggestive of focal lesion. Thirdly, we come to the hypothesis of the existence of one or other of those classic degenerations with which the neurologist is familiar. If infection, historrhexis, and classic degenerations can be excluded, we then proceed, fourthly, to the hypothesis of some kind of imbalance, perhaps metabolic or endocrine or sympathetic. If the diagnosis cannot be made on these lines, possibly the condition belongs in some miscellaneous and otherwise undefined or highly specialized group.

Even when the disease seems to be limited to a peripheral nerve, we consider that then the successive hypotheses of (1) infection, (2) historrhexis, (3) specialized neuronatrophy, (4) imbalance, can be preferably considered in that order. We think that by the pursuit of some such method as this the neurologist can bring his work better into line with that of general medicine. The method is, moreover, a very pragmatic method, since lines of treatment are specially indicated for the different great groups of disorders. But in so difficult a field we do not wish to dogmatize, and shall be content if our communication arouses interest in the application of the key or order principle to the diagnosis of nervous diseases.

THE FUNCTIONS OF THE CEREBROSPINAL FLUID

WITH A SPECIAL CONSIDERATION OF SPINAL DRAINAGE AND OF INTRASPINAL INJECTIONS OF ARSPHENAMIZED SERUM *

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THE PROBLEMS PRESENTED

The cerebrospinal fluid has of late years acquired a peculiar importance. This is true not only because of the rôle it has assumed in neurologic diagnosis, but also because of the various attempts that have been made to utilize it as a vehicle of therapeutic administration, or rather, because of the various procedures that have been evolved to use the subarachnoid space and the other containers of the cerebrospinal fluid as avenues of medication. Perhaps it will not be out of place to review some of the facts in regard to its physical and chemical constitution; in regard to its location and distribution; in regard to the laws of physics which it must obey; in regard to its source, it renewal and its escape; in regard to its relation to the vascular and lymphatic systems; in regard to the pressure under which it exists, and, finally, in regard to its movements and other cognate factors. To me it has seemed that the rôle and function of the cerebrospinal fluid has been strangely misinterpreted, and that this has led to attempts at therapeutic administration which may be justly characterized as unscientific; which have not been without danger, and which have either failed of their object or have owed such success as seemed to attend their application, to other and purely incidental factors which have been overlooked; this I hope to show. In any event, a consideration of the cerebrospinal fluid appears to me to be a very timely topic.

SIGNIFICANCE OF THE CHEMICAL COMPOSITION OF THE CEREBROSPINAL FLUID

When we turn our attention to the chemical composition of the cerebrospinal fluid, we are at once impressed by its resemblance to the ordinary three quarter per cent. common salt solution of the histologic laboratory. According to Halliburton,[1] it consists of water, inorganic salts similar to those of the blood plasma, a trace of coagu-

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* Read before the Philadelphia Clinical Association, Dec. 8, 1919.

[1] Halliburton, W. D.: The Possible Function of the Cerebrospinal Fluid, Brain, **39**:214 (Oct.) 1916.

lable protein and a certain amount of glucose. It is practically free from formed elements. Halliburton speaks of it as an "ideal physiological salt-solution," "as the perfect physiological medium," more perfect doubtless than the artificial fluids we can make in the laboratory, but in its essential features closely resembling those associated with the names of Ringer and Locke.[2] Mestrezat[3] has pointed out that the cerebrospinal fluid is identical with the ocular and labyrinthine liquids, and he has also emphasized the fact that the purely physical rôle of the latter is altogether indisputable. The liquid of the internal ear communicates directly with the subarachnoid space by means of the aqueduct of the cochlea, and it is proper to infer that it has no other origin; the mediums of the eye are independent, but they are beyond doubt formed by the ciliary processes which appear to be in every way comparable to the choroid plexuses. Mestrezat concludes that the cerebrospinal, the ocular and the labyrinthine fluids form a homogeneous family of liquids possessing a constitution almost exclusively mineral. They are liquids of "remplissage," i. e., of space filling, and of support and protection, and form a group of liquids by themselves. They owe their physiologic properties in part to their perfect fluidity which enables them to penetrate into the smallest interstices. The tonicity of the cerebrospinal fluid, as pointed out by Bard,[4] is constituted almost exclusively by the amount of contained sodium chlorid. This constitutes a negative property, i. e., a property which insures the perfect innocuousness of the liquid to the living elements with which it is in contact.

HYDROSTATIC PROPERTIES

Further, immersed in this salt solution, the brain is supported and buoyed so that its weight is relatively diminished. Mestrezat has calculated that the specific gravity of the cerebrospinal fluid being about 1.0075, and that of the brain 1.032 (women) to 1.041 (men), the pressure which the weight of the brain exerts on its base is reduced to the very low figures of from 26 to 39 grams. From these facts we may infer that a brain weighing from 45 to 50 ounces exerts a pressure of from about three fifths of an ounce to 1¼ of an ounce on its base.

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[2] Locke's fluid contains besides water and common salt, minute quantities of potassium chlorid, calcium chlorid, sodium bicarbonate and glucose. See Halliburton, Handbook of Physiology, 14th Ed., London, John Murray, 1919.

[3] Mestrezat, William: Le liquide céphalo-rachidien normal et pathologique, Paris, A. Maloine, 1912.

[4] Bard, L.: Le rôle de la pression dans l'action physiologique du liqueur céphalo-rachidien. Ses rapports avec les fonctions des plexus choroides et de la glande pinéale, J. de physiol. et de path. gén. **17**:171, 1917.

Further, this pressure is distributed over a relatively wide area. By means of this hydraulic suspension — for such it is — not only do the vessels of the base escape all compression from the weight of the brain, but the brain itself escapes the effects of pressure on its surfaces of support. Flattenings, deformities and distortions are prevented by means of this hydraulic suspension.

This hydraulic suspension and the relative loss of weight of the brain leads to another important result: The force of the impact of the brain on the hard cranial walls is diminished, i. e., the force of the blow which the brain itself, under given conditions, strikes against the cranial walls. I am speaking of such impacts as result not only from the numerous jars, shakings or concussions to which the head is subjected normally by the mere physiologic displacements of its position and by the movements of the body, but also of those more severe impacts to which accident exposes the skull and from which, short of actual surgical trauma, the brain remains uninjured. Further, as a result of the law of physics governing the incompressibility of liquids, the effect of impacts and concussions is diffused over the entire cranial contents, and blows and sudden displacements which might otherwise be followed by serious injury, if limited to a restricted area, have their force thus enormously diminished.

The softness and fragility of the substance of the brain is such that a very special protection against traumatic influences of all kinds, displacements and concussions, is absolutely necessary; hence we have a cavity with hard walls, practically a solid box, in the interior of which the brain is in a condition of hydraulic suspension. The changes which the movements of the head impose on the various surfaces of the brain render a hydraulic suspension especially necessary; the latter, let us bear in mind, exerts its action independently of position.

Liquids transmit pressure equally in all directions; notwithstanding, the gross weight of a column of liquid is in relation, of course, to its height. If the column is placed in a horizontal position there is no inequality of pressure exerted by its weight in any portion of the tube, while in the vertical position the pressure increases as we pass from the upper levels to the base. Changes of pressure must ensue, therefore, in the various levels of the cerebrospinal cavity in variations from the erect to the horizontal position. This fact is in accord with the notable difference observed in the pressure of the cerebrospinal fluid in making lumbar puncture, whether the patient lies down or sits up. It is a legitimate inference, therefore, that the column of cerebrospinal fluid in the spinal canal must in the erect position lessen relatively the pressure within the cranial cavity. As a result of the laws of hydrostatics, the pressure existing in the upper levels, say at the vertex, must be equal to the pressure existing in the lumbar region less the height of the column which separates the two points. Lessening the pressure in the lumbar region by spinal puncture, must therefore act in a decompressive manner comparable to that of an aspiration, a fact again in keeping within clinical experience. We should bear in mind, however, that in various pathologic conditions, differences of pressure in the levels of the cerebrospinal cavity may be much less than we would be led normally to expect; a fact which, when encountered, as in the instance of a subnormal intraspinal pressure, has in its turn a pathologic significance. The significance of an increased intraspinal pressure will be discussed later. All things considered, however, it should be stated that even under normal conditions the variations in the intraspinal pressure resulting from changes of the position of the cerebrospinal axis from the horizontal to the vertical are much less than we would *à priori* be led to suppose. Possibly, as Bard thinks, this points to some compensatory mechanism for the maintenance of a mean of pressure.

DISTRIBUTION, SOURCE AND EXIT

Leaving for a time the question of pressure, let us consider briefly the distribution of the cerebrospinal fluid, its source or sources, and finally its way or ways of exit from the cerebrospinal cavities. The cerebrospinal fluid is found in the ventricles and in the subarachnoid spaces. The subdural space, if it can be spoken of as such, is bounded by the two apposed moist surfaces of the dura and the arachnoid. It would appear that little if any interchange of fluid takes place between the subdural and the subarachnoid spaces, and further that if there be any passage of fluid it is from the former to the latter. Quincke,[5] for instance, injected cinnabar into the spinal subarachnoid space; none of the cinnabar found its way into the subdural space. However, injected into the cerebral subdural space, it found its way into the subarachnoid space. The pathway apparently was through stomata between the endothelial cells. I believe it probable that under normal conditions no exchange of fluid ever takes place. The arachnoid membrane is distended — ballooned as it were — by the pressure of the subjacent cerebrospinal fluid, and is thus closely apposed to the surface of the dura. Any lesion or break of its continuity would mean a failure of its function.

It is commonly accepted by physiologists and morphologists that the cerebrospinal fluid has its origin mainly in the choroid plexuses of the lateral ventricles, thence it passes through the foramen of Munro prussian blue granules were forced into places between which and the subarachnoid space no normal communication exists? And if this be true of the neuroglia may it not be true of the perivascular, the pericapillary and the perineuronal spaces as well? Finally, even admitting the validity of both Spina's and Weed's experiments, they would merely indicate the passage of material under certain conditions from the subarachnoid space into the perivascular spaces, and not from the perivascular spaces into the subarachnoid space; and it may be safely maintained that no evidence exists to prove the passage of material in the latter direction. We have just seen that while communications exist between the subdural space and the subarachnoid space, this communication is in only *one* direction, namely, from the subdural space into the subarachnoid, but not in the reverse direction. Surely in the present instance it is unjustifiable to assume, because under abnormal conditions pigment granules can be forced into the perivascular and perineuronal spaces and also into the neuroglia, that therefore a passageway normally exists and a flow normally takes place from these spaces into the subarachnoid spaces. To the consideration of this subject I will return a little later.

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[5] Quincke, cited by Weed, T. H.: J. M. Res. **26**:21, 1914-1915.

The principal source of the cerebrospinal fluid is, as we have seen, ascribed by physiologists to the choroid plexuses. If, as I believe probable, drainage material from the perivascular spaces is to be excluded, the question arises as to whether there are any other sources of the fluid. Obviously, the membranous surfaces suggest a source of supply. Physiologists in general are inclined to ascribe to these surfaces a very insignificant rôle. However, analogy would suggest a degree of such function on their part. We have merely to call to mind the synoviae of the joints, the peritoneum, the pleura, the pericardium and other serous surfaces. In the instance of the subdural space, the facts justify the inference that the fluid present, though small in amount, is not derived from the subarachnoid space. We again call to mind the exceedingly small avenues of communication between the ventricles and the subarachnoid spaces of the brain and spinal cord. It certainly seems strange that the large amount of fluid in the various cisternae and especially in the spinal canal should all find its way through the foramina of Luschka and the apochryphal foramen of Magendie. Further, a fact to which Cushing also calls attention[10] is that the ventricular and spinal fluids are not exactly identical in composition; for the ventricular fluid contains a higher percentage of sugar than the spinal fluid, while the spinal fluid contains a higher percentage of globulin. It is difficult to account for these differences by a single source of supply. Is it not possible that the rôle which the membranes play is greater than that commonly supposed? When we call to mind the occasional behavior of other serous surfaces — it is true under pathologic conditions — the idea does not seem so very improbable; and while the arachnoid itself has no vascular supply, its intimate neighbor, the pia, is richly endowed with vessels. Spina's experiments on the results of artificially induced cerebral hyperemia may have here a special significance.

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[10] Cushing, H.: J. M. Res. **26**:1, 1914-1915.

The next question that presents itself is as to the way in which the cerebrospinal fluid makes its exit from the cerebrospinal cavity. The admirable researches of Weed have shown that the cerebrospinal fluid makes its exit, in very large part, by means of the arachnoidal villi into the sinuses, i. e., into the venous system of the dura. The arachnoidal villi are extremely delicate structures which project into or are in close relation to the venous lacunae or with the lumen of the sinuses. As to the method of transition, Weed states that it becomes evident in the study of the mesodermal cells, which exist between the myxomatous groundwork of the villus and the lumen of the sinus, that the escape of fluid from the villi is probably through the cell substance. Weed uses the term "filtration" in describing the process; while this term appeals to me because it implies a purely physical process, it is perhaps objectionable on the ground that a filter removes or holds back something from a liquid undergoing filtration, and there is nothing in the functioning of the arachnoidal villi to indicate such a process. Perhaps the word osmosis or diffusion, both of which Weed mentions, would be more applicable; or better still, perhaps the word absorption would meet the facts best, as it is really a process of absorption of the cerebrospinal fluid by the venous blood. Weed also concludes, and in this he is in accord with other investigators, that in addition to the major return of cerebrospinal fluid by the arachnoidal villi, there is also an accessory drainage of fluid into the lymphatic system, though he believes that this plays a comparatively insignificant part in absorption except from the isolated spinal subarachnoid space; further, he concludes that absorption from the cranial subarachnoid space is much more rapid and much greater in amount than from the spinal portion. He also concludes, and this is most important, that no evidence has been afforded, in his observations, of the escape of cerebrospinal fluid into the cerebral veins or capillaries.

The lymph channels which aid in the escape of the cerebrospinal fluid are those in the sheaths of the cranial and spinal nerves. As just stated, these channels are mainly active in the spinal subarachnoid space, though there is no good reason for denying them this rôle in the cranial subarachnoid space. Perhaps in the spinal region they are the sole means of exit. According to Halliburton, diffusion of the cerebrospinal fluid is most rapid in the subcerebellar district and extremely slow in the spinal, especially in the lower spinal region. Halliburton also states, and in this he is likewise in accord with others, that diffusible substances introduced into the subarachnoid space pass rapidly into the veins.[11]

The last mentioned fact is of enormous practical value, for it shows that attempts to medicate the central nervous system, as is done especially in the intraspinal injections of arsphenamized serum in the Swift-Ellis method, are essentially unscientific and unphilosophical. The exceedingly minute — indeed, apochryphal — amount of arsphenamin contained in the serum is speedily removed by the dural sinuses and the lymphatic channels of the nerves, and never enters the nerve tissue. Still further diluted and weakened by the spinal fluid, it can at most pass over the membranes in its journey of exit. That the benefit which is supposed to result from the method is really due to another and entirely incidental factor will, I believe, become apparent as we proceed.

RELATIONS TO THE VASCULAR AND LYMPHATIC SYSTEMS: QUESTION OF NUTRITION OF NERVE TISSUE

Let us now turn our attention once more to the relation of the cerebrospinal fluid to the vascular system and also to the lymphatic system. We have already learned that the cerebrospinal fluid is in direct relation with the venous system through the arachnoidal villi and the dural sinuses; also, that it is in direct relation with the lymphatic system of the cranial and spinal nerves. What is its relation to the perivascular and perineuronal spaces? I have already advanced reasons for denying the presence of the communication assumed to exist between these spaces and the subarachnoid space, and denying them the rôle of being merely one of the cerebrospinal fluid containers. Let us for a moment call to mind some of the elementary facts of structure and physiology. Everywhere, in all of the organs and tissues, whether we think of the Haversian canals of the bones, or of the lymph sheaths which surround the blood vessels in the muscles, or of any of the other structures, we find that the nutrition of the tissues takes place not directly from the blood vessels, the capillaries, but through the intermediation of lymph channels. As Halliburton[11] expresses it:

> The lymph acts as the intermediary or middleman between the blood and the tissue elements, conveying to the latter on the one hand the oxygen and the nutritive substances they need; and on the other, it is into the lymph primarily that the tissues pour the waste products of their activity.

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[11] Halliburton, W. D.: The Possible Function of the Cerebrospinal Fluid, Brain **39**:214 (Oct.) 1916.

Why should we deny this rôle to the perivascular, pericapillary and perineuronal spaces? Weed sees an objection to considering these spaces as lymph spaces, saying:

> If this were the case, we should expect a content in lymph — cellular and containing many coagulable elements. The protein of such a tissue juice of lymph should suffice to keep patent the perivascular spaces after fixation for microscopic work. Ordinarily, however, these spaces are not distended, indicating clearly that the coagulable elements are very slight.

I would answer that these appearances are exactly those that are encountered in conditions in which the metabolism of the nervous tissue is increased, as occurs in certain pathologic conditions; notably in paresis in which even in relatively early stages the perivascular spaces are found enlarged and containing many formed elements. Further, the changes that take place normally during the process of nutrition in the lymph spaces surrounding the blood vessels of the various tissues are not such as are revealed by the microscope. The tissues receive their nourishment not from proteins in the lymph spaces but from amino acids which are the products of the cleavage of the proteins circulating in the blood vessels. In fact, this is true of all of the substances concerned in nutrition unless it be the mineral salts. The fats are split into alcohol and fatty acids, complex carbohydrates are fragmented, albumin is split up into peptones, the latter are reduced to amino acids and the amino acids into still simpler components. I submit, again, that such changes are not revealed by the microscope. Further, to include the perivascular spaces in the general cerebrospinal fluid space, of necessity, makes the content of the perivascular spaces the same as the content of the subarachnoid space and the content of the ventricles; in other words, it fills the perivascular spaces with cerebrospinal fluid. However, complex chemical changes, such as are concerned in nutrition, do not and cannot go on in the cerebrospinal fluid. The cerebrospinal fluid contains no ferments and can develop no ferments. It will not mend matters to say that the physicochemical changes of nutrition take place in the blood plasma and within the blood vessel walls, for the cells of the various tissues are the active agents which reduce — that is, split up — the materials offered by the blood plasma into components adapted to and peculiar to their individual structure; but between the cell and blood plasma lies the lymph space. Obviously, the change must go on through the lymph space; it cannot be otherwise. Surely, if the cerebrospinal fluid is the nutrient fluid of the brain, it should bear in its constitution some evidence of this fact.

Speaking of the perivascular spaces, Weed thus expresses himself:

> This whole accessory fluid system of the cerebrospinal canal axis—an intramedullary canalicular system—undoubtedly possesses an active function in maintaining the metabolic exchange and elimination of the nerve-cells. Throughout the body in the other tissues there is a chief and accessory circulation. For in these other tissues of the body there is in addition to the blood capillary a lymph capillary with the tissue juice or plasma playing the intermediate part in exchange.

Notwithstanding, in keeping with the long prevalent — and to my mind, erroneous—belief, he declares that "nervous tissue lacks entirely the lymphatic system, and that it would appear that its place is taken by the perineuronal, pericapillary and perivascular system." This, it seems to me, is a distinction without a difference, and it clearly has its origin in the mistaken view that the perivascular spaces share with the subarachnoid spaces and the ventricles the function of being cerebrospinal fluid containers. It is interesting to note in this connection that Mott,[12] in his Oliver-Sharpey Lectures on the Cerebrospinal Fluid, treats the perivascular space as a lymph space, though he believes that it communicates with the subarachnoid space. Speaking of the perivascular space, he says:

> It is filled by a *clear and transparent fluid* which from the point of view of its morphological significance should be considered as lymph. It contains in variable amount lymph corpuscles, fatty granules and sometimes even drops of oil.

Mestrezat[13] likewise maintains that the perivascular spaces constitute a lymphatic circulation, and, further, that this lymphatic circulation consists of a true lymph distinct from the cerebrospinal fluid and that no mixture of the two fluids takes places.

Unfortunately, the erroneous view that the perivascular spaces are filled with cerebrospinal fluid has given rise to the entirely untenable position that the cerebrospinal fluid is the nutritive fluid of the central nervous system. This is the view held, for instance, by both Weed and Halliburton. Halliburton tries to overcome one of the obvious difficulties by declaring that the amount of protein necessary for the repair of the nervous tissues is not great, implying that the amount of metabolism in nervous tissue is exceedingly small. However, it is probably much larger than Halliburton would lead one to suppose. What is the meaning of the enormously rich vascular supply to the brain and cord, if it does not predicate a large amount of nutritive change? What is the meaning of the enormous mass of capillaries if it does not imply an active metabolism? Indeed, one might ask, what is the need of a vascular supply at all if the nutrition of the central nervous system is carried on by the cerebrospinal fluid?

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[12] Mott, F. W.: The Oliver-Sharpey Lectures on the Cerebrospinal Fluid, Lancet **2**:1, 79, 1910.

[13] Footnote 3, p. 188.

It cannot be but that the nutrition of the nervous system is in keeping with that which goes on elsewhere in the body, and that definite chemical and physical changes take place in it as in the other tissues. In muscle, for instance, the chemical reaction, which is neutral or feebly alkaline when the muscle is at rest, becomes acid in a muscle that is active; there is also in the active state a greatly increased elimination of carbon dioxid while the tissue consumes proportionately more oxygen; further, the active muscle contains more water, it yields an augmented quantity of extractives soluble in alcohol, a lessened quantity of extractives soluble in water, a lessened quantity of substances producing carbon dioxid, a lessened quantity of fatty acids and of creatin and creatinin, and a lessened quantity of glycogen. Its circulatory relations are modified; its capillaries dilate; it contains more blood, and the blood in its veins differs chemically from that found in its veins when at rest.

Nerve substance, like muscle, when at rest is neutral or feebly alkaline, and when active is acid. It has not, however, been possible to determine the chemical changes in detail. Not even the exchange of oxygen and carbon dioxid has been demonstrated, though the facts attending vascular supply and temporary obstruction justify the inference that such changes of necessity take place. The effect of temporary compression of the vessels of the brain or of the abdominal aorta in producing arrest of function — of the brain, on the one hand, or of the cord, on the other — is an instance in point. Halliburton[14] himself admits that the nerve centers are richly supplied with blood vessels which furnish them with an abundant supply of nutrient material, that cerebral anemia rapidly produces pathologic changes in the nerve cells, and that death quickly supervenes. Further, a number of observers have demonstrated that nerve cells at rest and in activity present marked differences histologically. Thus Hodge, Vas, Nissl, Mann, Lugaro and others have studied the changes in nerve cells that result from function. While the various investigators differ somewhat as to details, they agree as to the essential facts. They have discovered that during rest the chromatic substance in the cells increases in amount, and that during functional activity it diminishes. Activity of the nerve cell is first accompanied by swelling of the protoplasm of the cell body, later by a progressive diminution in the size of the cell body. If functional activity be prolonged, the nucleus undergoes changes similar to those of the cell body, and this is true also of the nucleoli; there is at first an increase in volume, which subsequently gives way to the reducing action of fatigue. Halliburton[14] tells us that cholin, derived from the decomposition of lecithin and other phosphorized fats, is obtained from perfectly fresh nervous tissues, that its presence is a very positive sign of chemical activity and that the gray matter yields the most cholin. Unfortunately, little is known of the chemical changes in the blood of the venous return; but certainly such facts as are in our possession are in favor of metabolic changes taking place in nerve tissue of no mean degree of activity. This is also in keeping with the enormous activity of the nervous centers which, with the possible exception of those portions concerned in consciousness, is incessant. To me the conclusion is more than justified that the chemical changes taking place in the central nervous system are, to say the least, considerable, and that they take place not through the cerebrospinal fluid but through the blood vessels and in the spaces which surround the latter, and that the function of these spaces is indistinguishable from the perivascular lymph spaces existing elsewhere. These considerations are, as we will see, not of theoretical interest purely, but of great practical importance. Finally, a conclusive proof that there is no communication between the perivascular, pericapillary and perineuronal spaces on the one hand, and the cavities that contain the cerebrospinal fluid on the other, is furnished by the fact that the cerebrospinal fluid normally contains no products of nerve tissue metabolism. Halliburton[15] tells us that normally it contains no cholin, no cholesterol, no nucleoprotein.

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[14] Halliburton, W. D.: Lectures on Biochemistry of Muscle and Nerve, Philadelphia, P. Blakiston's Son & Co., 1904, p. 79.

PRESSURE RELATIONS: SPINAL DRAINAGE

Let us now turn our attention to the pressure under which the cerebrospinal fluid exists; the relations of this pressure to that of the arteries, capillaries and veins, and to that in the dural sinuses; also to the movements of the cerebrospinal fluid, and, finally, to questions of its renewal, all of which questions have a practical bearing.

We have already considered in part the question of the pressure of the cerebrospinal fluid in relation to its hydrostatic properties. Studies and estimates of the pressure of the cerebrospinal fluid have been made by a large number of observers. The figures that are given disclose a wide range of variation; for example, Cybierski would place the pressure at from 72 to 90 mm. of water, while Bard would place it at 200 mm. Perhaps it is fair to conclude, as does Richet[16] from averaging the results of Quincke, Krönig, Parizot and Boveri, that it is about 100 mm. Doubtless the differences in figures are due in large part to differences in the methods of investigation employed. For us, however, the fact of physiologic importance is that the cerebrospinal fluid normally exerts a pressure on the walls of its containers, and that this pressure is greater than that in the venous sinuses; absorption by the latter of the cerebrospinal fluid through the arachnoidal villi is thus directly favored. The pressure of the cerebrospinal fluid is also greater than that in the lymphatics,[17] which fact likewise favors the theory of absorption. It should be added that Bard estimates the pressure in the cerebral capillaries to be six or seven times as great as that of the cerebrospinal fluid; so that it is extremely improbable that any absorption takes place into the capillaries as is supposed by Mott. The pressure in the cerebral arteries is also notably higher than that of the fluid; that in the cerebral veins is clearly higher than that in the sinuses. The cerebral arteries, capillaries and veins, it should be borne in mind, have no relation of absorption with the cerebrospinal fluid. The relations of the latter are merely with the sinuses and with the lymphatics of the cranial and spinal nerves.

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[15] Halliburton: Handbook of Physiology, 1919, p. 168.

[16] Footnote 7, p. 53.

The pressure of the spinal fluid is doubtless maintained by a continuous supply. The latter is furnished as we have seen by the choroid plexuses[18] though other sources are not excluded. The questions arise,

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[17] Footnote 7, p. 54.

[18] Bard (Footnote 4), after commenting on the numerous plications of the choroid plexuses, estimates the surface of the latter as equivalent to 1 square meter.

Bard thinks that the vascular richness of the plexuses permit of great variations in volume and that this exerts beyond a doubt an influence on the pressure of the liquid in which the plexuses are immersed. He is led to think, further, that the regulation of the spinal fluid pressure may depend on two different mechanisms, one for changes slow and persistent, and another for changes sudden and rapid, such for example as are caused by changes in attitude. The first depends on the quantity of cerebrospinal fluid present, as determined by the relative rates of formation and absorption. The second, Bard thinks, is a function of the pineal gland which he has come to regard as a barometric perceptor and regular. Its action would seem to influence the distention and activity of the choroid plexuses. The discussion of this interesting subject would, however, take us too far afield.

Bard also points out that the pressure of the cerebrospinal fluid is in relation with the external atmospheric pressure through the round window of the internal ear, and suggests that perhaps changes in the pressure of the fluid could be clinically studied in the ear, thus rendering a spinal puncture unnecessary. Personally it is not clear to me how the round window could be observed. However, the pressure of the fluid is also in relation with the oval window. Is it possible, perhaps, that transmitted through the chain of bones, changes of pressure could be studied through the drum membrane?

What is the total amount of cerebrospinal fluid and how often is it renewed? The total amount of cerebrospinal fluid has been estimated at from 125 to 150 c.c. It appears to be undergoing a gradual and constant renewal. Mestrezat, Frazier and others believe that it renews itself six or seven times in twenty-four hours. I am inclined to think that this estimate is excessive, and that normally the spinal fluid renews itself much more slowly. The flow of cerebrospinal fluid observed every now and then in fractures of the cranium, in disease of the ethmoid, and in other pathologic states in which the subarachnoid space is in communication with the external world — a flow which is sometimes extraordinary in amount — must not be taken as the measure of the fluid normally produced, but should be looked on as an effort on the part of nature to maintain the normal degree of pressure by an excess of secretion, i. e., an effort to overcome the leakage. This fall of pressure caused by the leakage results, it would seem, automatically in an increased secretion. This fact — namely, of a fistulous opening greatly stimulating the formation of cerebrospinal fluid — has an important bearing on the question of a therapeutic expedient which has for a number of years past been practiced extensively in my clinic at the Jefferson Hospital, namely, spinal drainage. The flow which follows a lumbar puncture is at first very free; very soon the flow moderates and the fluid escapes from the cannula by a series of drops. Gradually these become less frequent until finally the fluid escapes in drops formed at the end of the cannula at intervals of several seconds, five or longer. It is interesting to observe that the final rate no longer changes, but is steadily maintained, no matter how long the cannula remains in position. In other words, the puncture has not only permitted the escape of fluid in an amount equivalent to a fall of pressure equal to that of the atmosphere, but has also, and by virtue of this very fact, stimulated the production of a fresh amount of fluid. In this way spinal drainage results, when practiced thoroughly, in a veritable washing out, a lavage, of the subarachnoid space.

MOVEMENTS: MAINTENANCE OF PRESSURE: NATURE OF THE FLUID

Before considering spinal drainage in detail and in order to complete our study of the functions of the cerebrospinal fluid, let us briefly consider its movements, the causes of the maintenance of its pressure, and, finally, its nature. It is exceedingly probable, as already pointed out (page 234), that the cerebrospinal fluid passes from the cranial subarachnoid space into the spinal subarachnoid space. Does it remain there to be disposed of by gradual absorption by the lymphatics of the spinal nerves, or is there a return flow into the cranial cavity, and does the one time spinal fluid share in the exit of the cranial fluid through the arachnoidal villi? If there is a return flow, what is the pathway? These questions cannot be definitely answered. The difference between the chemical constitution of the cerebral and spinal fluids, which has already been commented on, though slight, is opposed to a free commingling of the fluids such as would be suggested by an actual return flow. It is true, as has already been stated, that, under experimental conditions in animals, pigment granules injected into the spinal subarachnoid space find their way into the cranial cavity, but the condition of the experiments, as has been pointed out, are such as to lend no justification to the inference that such a passage actually occurs normally.

We are all familiar with the phenomenon of brain pulsation, synchronous with the heart's action, a pulsation visible to the eye on the exposure of the brain at the time of operations, and also palpable in the fontanels of the infant and in adults in whom a portion of the cranium has been lost. What happens in the closed cranium at the time of a systole? Both the cerebrospinal fluid and the blood being liquids, they must obey the physical law of incompressibility. Something, however, must yield. What is it? Bard believes that at the moment of systolic arrival of the arterial blood, a pulse wave is created in the cerebrospinal fluid as a result of which cerebrospinal fluid flows — is driven — into the vertebral canal, that this outflow compensates for the inflow of blood, and that this is followed during diastole by a flow of the fluid in the reverse direction. He states that the outflow of the fluid has been demonstrated by François-Franck by placing a hemodrometric pallet in the occipito-atloidian space. Cathelin disputes the reality of this oscillation, and claims that the outflow of fluid alone takes place and that the fluid leaving the cranial cavity continues its exodus without turning backward. It is a little difficult to accept the theory that at the moment of systole, cerebrospinal fluid is driven from the cranial cavity into the spinal cavity. In the first place, the latter is already full and its walls can hardly be said to be more distensible than those of the cranial cavity, and, further, its arteries are also conveying blood into the spinal cavity at the same moment that blood is entering the cranial cavity. Is it not more likely that of the contents of the cranium, it is the tissue of the soft nonresisting nervous system which in some way adapts itself to the increased pressure of the arterial wave? This I believe is the only possible explanation. Brain tissue as a whole is readily compressible, and probably the increase of pressure caused by entrance of blood at the moment of systole is accompanied by a yielding — not on the part of the neurons and nerve fibers — but by a yielding on the part of the perivascular spaces and perhaps in a degree by a yielding of the soft neurogliar tissue. Two things would result from such a mechanism: first, the blood, itself incompressible, would be driven into the soft tissue of the brain, the latter immersed in an incompressible medium, the cerebrospinal fluid, with relatively great force. In other words, the mechanism by means of which the brain tissue is placed between two incompressible mediums would result in the capillaries being very thoroughly filled at each systolic wave. Second, it is exceedingly probable that the contents of the perivascular spaces are gently moved onward at the same time; besides, the perivascular spaces afford the space necessary for the play of the expanding and contracting blood vessels. That the perivascular spaces probably terminate, as do the perivascular lymph spaces of all other tissues, in channels in or about the walls of the blood vessels and perhaps in other ways not yet recognized, and so make their exit from the cranium, is to my mind exceedingly probable.[19]

Respiratory movements, as is well known, also influence the movements of the intracranial contents, a fact which is familiar to every one who has practiced lumbar puncture. The respiratory pulse wave, however, makes itself evident only through the intermediate action of the vascular apparatus and the problems presented in no wise differ from those just considered and need not detain us.

What are the causes of the maintenance of pressure of the cerebrospinal fluid? Evidently these must be sought in the continuous formation of the fluid. I have already commented on the rôle played by the choroid plexuses. There has been a tendency of late to speak of the choroid plexuses as the choroid "glands," and of the cerebrospinal fluid as though it were a glandular secretion. But is it proper to speak of it in this sense? A glandular secretion is a fluid containing new bodies, new chemical principles, ferments, hormones or other substances not previously existing in the blood, but which have been elaborated from the blood plasma by the specific action of glandular cells. Assuredly, the cerebrospinal fluid presents no such constitution. Indeed, as we have seen, it presents little more than the three quarter per cent. common salt solution of the histologic laboratory. Mestrezat, because of the inorganic constitution of the cerebrospinal fluid, terms it a "mineral serum" and contends that its constituents exist preformed in the blood plasma and that they are derived from the latter by a

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19. Admitting for the sake of argument that the perivascular spaces are really cerebrospinal fluid containers, the question arises, Do these spaces leave the blood vessels at some hypothetical point or points to enter, like so many tubes, the subarachnoid spaces? and if so, Are the blood vessels distal to such point or points, either in the nerve tissue or in the pia, devoid of spaces—lymph or other—about or in their walls? Such a supposition is obviously absurd and we have here another reason for regarding the perivascular spaces as being related intrinsically and essentially to the blood vessels.

process of dialysis pure and simple through the epithelium of the choroid plexuses. Lochelongue,[7] it should be added, also holds to the view that the cerebrospinal fluid is not a true secretion.

ACTION OF THE CHOROID PLEXUSES

It would appear that the action of the choroid plexuses, and more particularly of their investing epithelium, is to permit the passage into the cerebrospinal fluid of water and of such a minute quantity of salines and other substances as are necessary to preserve the innocuous and neutral character of the fluid, and also its specific gravity, properties so necessary to its service as the medium of hydraulic suspension of the central nervous system. The passage of all other substances is inhibited or prevented. The inability of various substances thrown into the circulation to find entrance into the cerebrospinal fluid, a fact demonstrated by Frazier[9] and others, is well known. This fact, however, does not mean that it is the function of the choroidal epithelium and of the lining mesothelium of the subarachnoid spaces to prevent toxic substances from reaching the central nervous system, but merely to *prevent such a change in the composition of the fluid as would interfere with its action as the innocuous medium of hydraulic suspension.* Bard characterizes the choroidal epithelium as a simple barrier which prevents little else than water and sodium chlorid from passing through.

UNSCIENTIFIC CHARACTER OF INTRASPINAL MEDICATION

The mistaken view that the nutrition of the nervous system is carried on by means of the cerebrospinal fluid has led to attempts to utilize the cerebrospinal fluid of the spinal subarachnoid space as a medium of medication. Noticeably has this been the case in the treatment of paresis and tabes by intraspinal injections of so-called arsphenamized serum, the Swift-Ellis method. The belief has been that by this means the obstruction which the choroid plexus is supposed to offer to the arsphenamin could be circumvented. I have already pointed out that substances introduced into the cerebrospinal fluid rapidly disappear, passing through the arachnoidal villi into the venous system without entering the parenchyma of the central nervous system in the slightest degree. If arsphenamin acts at all when introduced in simple solution into the spinal subarachnoid space, it is by irritating and inflaming the spinal membranes, and, in a variable degree, the other spinal contents. This is the legitimate inference to be drawn from the experience of Paul Ravaut[20] and of others; unless at least the arsphenamin has been used in such small amounts as to be under any circumstances practically valueless. These conclusions doubtless apply with equal force to the introduction of corrosive sublimate and other antisyphilitic remedies via the subarachnoid route.

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[20] Ravaut, P.: Ann. de méd. **1**:49, 1914.

I am, of course, well aware of the claims made in behalf of the Swift-Ellis and allied methods, especially in the treatment of tabes, of spinal syphilis of the exudative form, and to a less extent of paresis. For a long time I made use of this method most extensively in my clinic and in my private work, and I can confirm the claims made as to undoubted improvement in tabes and spinal syphilis, and of apparent improvement from time to time in paresis. I have, however, for several years past come to the conclusion that the improvement does not depend on the intraspinal injection of the arsphenamized serum, but on the drainage of the spinal subarachnoid space which must inevitably be practiced before the injection is made.

SPINAL DRAINAGE: ITS MODE OF ACTION

Several years ago, Dr. Sherman F. Gilpin, chief of my clinic, conceived the idea that it would be a good plan to practice spinal drainage at the time of giving arsphenamin intravenously in the hope that, the intraspinal pressure being reduced, the arsphenamin circulating in the blood would be more likely to diffuse into the subarachnoid space. Later, Dr. Gilpin together with Dr. Thomas B. Earley instituted simple spinal drainage alone. The results were striking. Marked improvement followed, an improvement which was more pronounced than that observed in the Swift-Ellis method, doubtless because the drainage was more thorough. The improvement was most decided, as might have been expected, in tabes, but was also very evident in paresis. The patients are put to bed and drained of every possible drop of fluid about once in a week, once in ten days, or once in two weeks. The mode of operation of the procedure is probably as follows: To begin with, there is an immediate reduction of the increased intradural pressure; secondly, the drainage results in a kind of lavage of the dural space, for the spinal fluid which has been removed is rapidly replaced. In keeping with this fact, there is a fall in the lymphocyte count. Further, there is normally a balance between the pressure in the blood vessels within the cord and the pressure of the surrounding cerebrospinal fluid. It follows that if the pressure of the cerebrospinal fluid is abnormally increased, the vascularity of the cord will be correspondingly diminished; less blood than normally will be able to enter its vessels. It would appear, therefore, that the rapid withdrawal of the cerebrospinal fluid is followed by an increase in the vascularity of the cord — a relative hyperemia — and other things being equal, of the brain as well. Probably the results — often truly remarkable — which follow radical spinal drainage are to be attributed to the improved nutrition following this increased vascularity. We have here a parallel to the results of the Bier method in surgery. It should be added that we have never noted any untoward results from spinal drainage in either paresis or tabes. That the diagnosis — previous to drainage — should definitely exclude brain tumor and brain abscess goes, of course, without saying. Finally, it is interesting to note that the reason the pressure of the cerebrospinal fluid is pathologically increased in tabes and paresis is that its outflow is obstructed. It would appear that the inflammation of the membranes, the meningitis, present in these affections leads to an occlusion more or less pronounced of both the arachnoidal villi and the lymphatics. The cerebrospinal fluid continuing to be formed, a rise of pressure necessarily follows. Spinal drainage is, therefore, directly indicated in both tabes and paresis.

For a long time past, we have abandoned the Swift-Ellis method in my clinic. We have relied altogether on intravenous injections of arsphenamin and mercurial inunctions together with spinal drainage, and our results have been better than previously, doubtless because we have given especial attention to the drainage, removing at each occasion all the spinal fluid possible. Finally, in a number of cases, we have practiced spinal drainage alone, and always with improvement.

It is on the incidental spinal drainage, practiced in the Swift-Ellis and allied methods, let me repeat, that such success as has attended the procedure depends, and not on the apochryphal amount of arsphenamin contained in the serum. I am entirely in accord with the position assumed by B. Sachs[21] as to the uselessness and unnecessary character of the method. Besides Sachs tells us that with Professor Benedict he has succeeded in showing that arsphenamin introduced in the usual quantities into the blood current appears in the cerebrospinal fluid in appreciable quantities. Sicard and Bloch[22] have likewise noted this fact, the arsenic making its appearance in the cerebrospinal fluid an hour after the intravenous injection. Kopke[22] also noted the presence of arsenic in the cerebrospinal fluid after the administration of atoxyl in trypanosomiasis. Even admitting, therefore, that the presence of arsphenamin in the cerebrospinal fluid is of any consequence, no pretext can any longer exist for the intraspinal injections of arsphenamized serum.

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21. Sachs, B.: On the Intravenous, Not the Intraspinal, Use of Arsphenamin in Syphilis of the Nervous System, Arch. Neurol. and Psychiat. **1**:277 (March) 1919.

22. Cited by Mestrezat (Footnote 3) p. 93.

THE REAL DIFFICULTIES OF ANTISYPHILITIC MEDICATION

The difficulty with the various antisyphilitic remedies at our command is not that when introduced by the mouth or parenterally, they do not pass through the epithelium of the choroid plexuses, but because they do not pass through the walls of the capillaries. The reason that the iodids, mercurials and arsphenamin are so efficacious in syphilis of the exudative, the gummatous, form, is because in this form the spirochete has invaded especially the lining membrane, the walls of the blood vessels and the immediately contiguous tissues. These structures are all of them directly accessible to the action of remedies circulating in the blood. The walls of the capillaries, however, offer an obstruction to their entrance into the pericapillary and perineuronal spaces. Therefore, no action by such remedies can take place on the nervous parenchyma. How seriously the latter is invaded in both paresis and tabes, we all know. It is here that the difficulty lies and not in the barrier offered by the choroidal epithelium. An element of hope is presented in that the walls of the blood vessels do not offer the same rigid barrier as does the choroidal epithelium so necessary to the preservation of the integrity of the cerebrospinal fluid. We know that the capillaries do permit various drugs to pass through; among these are the alkaloids; morphin; cocain; caffein; thein; quinin; various salts, such as the bromids; various narcotics, such as trional, sulphonal; various urea derivatives, such as veronal, medinal, luminal, besides urotropin and the volatile anesthetics; doubtless many other substances could be enumerated.[23] It would seem that the walls of the capillaries are especially impervious to substances containing the ions of the metals. Two things appear to be necessary to the conquest of parenchymatous nervous syphilis: one, an increased knowledge of the biology of the *Spirochaeta pallida,* and the other, the development of a diffusible and innocuous spirochetocide based on this knowledge. In other words, the problem is one of biology and biochemistry.

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[23] Halliburton writes, "If, as Weed has suggested, the cerebrospinal fluid is partly formed, as ordinary lymph is, by exudation from the blood into the perivascular spaces, it is difficult to understand why readily diffusible drugs and poisons do not escape readily into the cerebrospinal fluid as they do into ordinary lymph," Brain, loc. cit., p. 219. As the writer points out above, many drugs and poisons undeniably diffuse through the capillaries and the pericapillary and perineuronal spaces, and the reason they are not found in the cerebrospinal fluid is that these spaces do not—as is commonly supposed—drain into the cerebrospinal fluid. All difficulties disappear when this mistaken interpretation is abandoned.

CONCLUSIONS

Leaving this digression, let us briefly summarize the conclusions which the foregoing considerations of the cerebrospinal fluid appear to justify.

First, the cerebrospinal fluid is preeminently a fluid for the hydraulic suspension of the brain and cord; its function is essentially hydrostatic.

Second, its chemical constitution is essentially that of the innocuous three quarter per cent. common salt solution of the histologic laboratory. It has no action on the tissues with which it comes in contact: it is absolutely neutral and negative.

Third, it is distributed through the ventricles and subarachnoid spaces. It has no relation to the perivascular, pericapillary or perineuronal spaces.

Fourth, it possesses no function of and plays no rôle in nutrition. The nutrition of the brain and cord takes place as does that of the other tissues — through its blood vessels, the perivascular spaces playing the same rôle as do the perivascular lymph spaces in the other organs and tissues. The old belief that the brain and cord have no lymphatic system must be abandoned.

Fifth, the cerebrospinal fluid has its source in the choroid plexuses and perhaps in the general serous surfaces of its containing cavities. It leaves the subarachnoid spaces of the cranium by passing through the arachnoidal villi into the venous current of the sinuses; also to a lesser extent by the lymph sheaths of the cranial nerves; from the spinal subarachnoid space it passes out by the lymph sheaths of the spinal nerves.

Sixth, attempts at medication of the brain and cord through the subarachnoid space, as in the Swift-Ellis method, are unscientific, as substances introduced into the cerebrospinal fluid rapidly disappear by passing out through the arachnoidal villi and the lymph spaces without in the slightest degree penetrating the nervous parenchyma; the beneficial effects hitherto ascribed to the Swift-Ellis and kindred methods are due entirely to the incidental spinal drainage.

Seventh, medication of the nervous parenchyma must be attempted through the alimentary tract, through the skin, through the areolar tissue, or directly through the blood.

Eighth, a remedy should be sought the ions of which will readily osmose through the capillary walls.

Ninth, spinal drainage is urgently indicated in tabes and paresis for the reasons enumerated in this paper.

A CASE OF MYXEDEMATOUS PSYCHOSIS

CLINICAL AND PATHOLOGIC REPORT

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INTRODUCTION

In 1873, Gull described five cases of a peculiar disease under the title "On a Cretinoid State, Supervening in Adult Women," and explained the similarity to sporadic cretinism, which was described by Hilton Fagge. In 1877, Orth reported five similar cases, and noticed especially the condition of the skin, in which he found an excess of mucin. He, accordingly, named the disease myxedema. He found also, in a case that came to necropsy, atrophy of the thyroid gland, which was previously noticed by Curling in a case of sporadic cretinism.

An exact understanding of this condition, however, is dated from the time when Reverdin and Kocher recognized cachexia strumipriva, and when the same condition was brought out experimentally by removing the thyroid.

Semon is the first one who claimed definitely that myxedema, cachexia strumipriva, sporadic and endemic cretinism, are in the same disease group, and are characterized by the failure of the function of the thyroid.

Alcoholism, syphilis and numerous pregnancies, especially when accompanied with much loss of blood, have been thought to be the cause of myxedema. Kocher described a case in which myxedema was accompanied by gummatous syphilis of the thyroid, both being cured by iodin treatment. The same author observed another case in which actinomycosis was the cause of the disease; the patient was cured by incision and drainage. Sometimes myxedema is found in conjunction with goiter. These cases are reported by Murray, Heinzheimer, Gantier, Robertson, Imredy, Hischl, Ulreich and others. Among the most important causes of the disease are also acute infectious diseases, such as typhoid, malaria, acute rheumatism, pneumonia, etc.

Though the disease is found nearly everywhere, it is said to be more frequent in England, France and Switzerland. It occurs in cold climates rather than in the tropics, but it is not common in North America.

Women are far more frequently afflicted than men. Prudden found 145 cases, 32 men and 113 women; Murray, 425 cases, 55 men and 370 women; Heinzheimer, 127 cases, 10 men and 117 women. The predisposition of women is probably due to frequent congestions and toxic infectious disturbances of the thyroid during menstruation, pregnancy, menopause, etc.

The most pronounced symptoms are myxedematous infiltration of the body, dryness of the skin, falling of the hair, slowness of the movements and certain nervous and mental symptoms. Sensory organs are often afflicted; hearing, taste and smell are diminished. Deafness is quite common, and it is thought to be of central origin. The extremities, lips and nose are cold and cyanotic, circulation is slow and the pulse rate varies from 50 to 65. Cerebellar symptoms due to the disease were reported in 1910 and 1911 by Soederbergh.[1] He observed cases with adiadokocinesis, Babinski's cerebellar catalepsy and exaggerated irritability of the muscles, but without symptoms of dementia.

The frequency of myxedematous psychosis is differently reported by various authors. Wagner and Jauregg[2] gave a percentage of 15. In the majority of cases the psychosis occurred late, after many years' duration, but in a few cases the psychosis occurred in the beginning of the disease, as reported by Pilcz, Inglish and Sierau.

The symptoms of the psychosis are not characteristic. Most patients seem to be demented, with more or less delusions, which are not infrequently based on hallucinations. The delusions are apt to be persecutory in character. The tendency to misbelieve is claimed to be one of the characteristics of the disease, and is an important factor in the development of the delusional conditions. Under the influences of persecutory ideas, patients are apt to become disturbed, cry, become agitated and sometimes extremely violent. In other cases there are reported several different symptoms, such as grandiose ideas, manic exaltations, or melancholia, anxiety and suicidal ideas. The latter cases are reported by Pilcz, Kraepelin, Show, Inglish, Beadles, Clouston, Hamilton, McLane and others.

The most important pathologic anatomical finding is that of the thyroid; even by palpation the organ shows remarkable atrophy. At necropsy it weighs one-tenth of the normal gland. Burghart observed a case in which he could not demonstrate any gland tissue. Some authors, such as Cunningham, Robinson, Corkhill, Schwass and others, have reported cases of enlarged thyroid in living patients. Microscopic examination shows marked increase of fibrous tissue. Some authors observed lymphocytic infiltration in the parenchyma in the early stage of the disease. According to Prudden, the parenchyma of the gland is more or less completely replaced by fibrous tissue and by newly formed reticular tissue in which are lymphocytes, resembling the tissue of the lymph nodes.

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[1] Söderbergh: Ueber Pseudotetanus myxoedematoides, Monatschr. f. Psychiat. u. Neurol. **32**:402, 1912. Encore un cas de myxoedème avec symptômes cérébelleux, Rev. neurol. **19**:86, 1911. Faut-il attribuer à une perturbation des fonctions cérébelleuses certains troubles moteurs du myxoedème, Rev. neurol, **2**:487, 1910.

[2] Wagner and Jauregg: Myxoedema und Kretinismus, Handbuch der Psychiatrie, Aschaffenburg, 1912.

The majority of authors consider that the increase of the connective tissue is secondary, and that the primary cause is the atrophy of the gland tissue, which is probably due to some toxic agent. Ponfick believes in the possibility of a primary infection of the interstitial tissue which may give rise to secondary atrophy of the gland tissue.

The pituitary body is often found enlarged, as in the cases of Boyce and Beadles, Ponfick, Murray and others, but in one case Ponfick found atrophy of the gland. The relation of the pituitary body to myxedema still remains obscure.

No characteristic, regularly appearing findings have been reported in other organs of the body. The pathologic anatomy has given no satisfactory explanation for so marked disturbances in the mental and psychical realms. Brain, spinal cord and nerves are found either normal (Hun, Prudden, West and Harely), or the findings are not absolutely characteristic.

In about half of the cases atheroma of the aorta and large arteries has been found. Endarteritis of the small arteries is also one of the frequent findings.

In the Danvers State Hospital laboratory we have had two cases of myxedema in a total of 2,130 necropsies, beginning in 1879 and ending in the present year. The necropsy of the first case, a 45-year-old female, case number 15840, was performed (Nov. 16, 1910) by Dr. A. H. Peabody.[3] The clinical symptoms and pathologic findings correspond to those of a typical case. But, I regret to say, I could not study that case in connection with the present one, because the brain was not available and the description concerning the central nervous system is missing. Some of the clinical and pathologic symptoms, however, show striking similarity to the present case, and will be referred to later in the discussion. The second case is the present one, of which we have been able to make a thorough study and to report as a typical case of myxedema.

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[3] Peabody: An Histological Study of the Thyroid Gland in Mental Disease, with Special Reference to Chronic Thyroiditis, Danvers State Hospital Laboratory Papers, 1910.

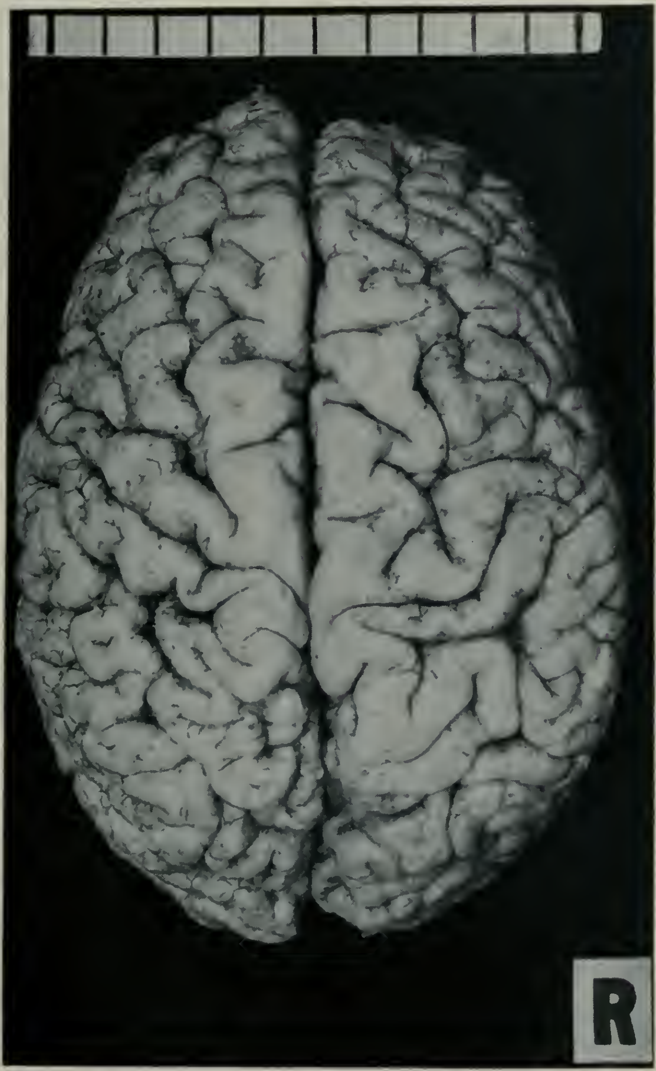

Fig. 1.—Superior surface of the brain.

REPORT OF A CASE

CLINICAL OBSERVATION

History (Danvers State Hospital, Clinical No. 21477).—There was no history of nervous or mental diseases in the family, so far as was known.

The patient was born Jan. 1, 1853, in Nova Scotia. Early development showed nothing abnormal. She had very little education, and did not go to school until she was 9 or 10 years old, and then only for six months. She could barely read and write. She left home at 18 to do housework, and when about 20 married her cousin, by whom she had two children, one of whom died in infancy. Her husband died of typhoid and pneumonia two years after marriage, and shortly after that she came to the United States (about forty years ago), and did general housework. Thirty-eight years ago she married again, and had three children, one of whom died in infancy. The informant stated that the patient had some pelvic trouble ten years ago when about 55 years of age, and was operated on for two large broad ligament cysts on the right side; at the same time her appendix was removed.

Since that time she had been gradually failing, both mentally and physically. Her memory, especially for recent events, began to fail. She had had many attacks of vertigo, sometimes falling to the floor. These symptoms have gradually been growing worse, especially during the past year. About a year ago she was in danger of losing her home, and her son took her to his home and tried to keep her with him. She had always been very stubborn, self-willed and dictatorial, and insisted on having her own way. He could do nothing with her, and during the past year her daughter has been taking care of her. She became very untidy, and would wander about on the streets attired very carelessly. She persisted in going back to her own home. She would put things away, lose them, and then accuse her daughter of stealing them. At times she seemed depressed. She threatened to commit suicide, and on one occasion she tried to turn on the gas. Her daughter was afraid to leave her alone in the house. She would meddle with the cooking, and spoil the food by putting salt in it; she was dull and stupid, sleeping a good deal during the day. Her appetite was capricious; sometimes she ate an enormous amount. She was admitted to the Danvers State Hospital, March 24, 1919.

Physical Examination.—General: Her height was 4 feet 8 inches; her weight, 130 pounds. She was well developed and well nourished. The skin over her face and neck was pale yellowish and waxy. The musculature showed poor development, not much tone. Edema of the face, arms and legs was pronounced. The hair was grayish and very sparse; she was bald over the vertex. There was a linear scar extending from the umbilicus to the pubic bone.

Thoracic, Respiratory and Circulatory Organs: Results of the examination were negative except that respiration was slow, 14 per minute, and the pulse rate was only 60 per minute. The body temperature on admission was 98.8 F.

Digestive and Abdominal Organs: Teeth were missing in the upper jaw, and a few stumps remained in the lower jaw. The breath was fetid. The tongue was extremely large and flabby and was slightly coated. The abdomen was large and pendulous.

Genito-Urinary Organs: No internal examination was made. The urine (March 27, 1919) was cloudy and straw colored. The specific gravity was 1.026. It was acid and showed a trace of albumin, mucous threads, epithelial cells, a few leukocytes and a few oxalate crystals, but no sugar. The Wassermann reaction on the blood serum was negative.

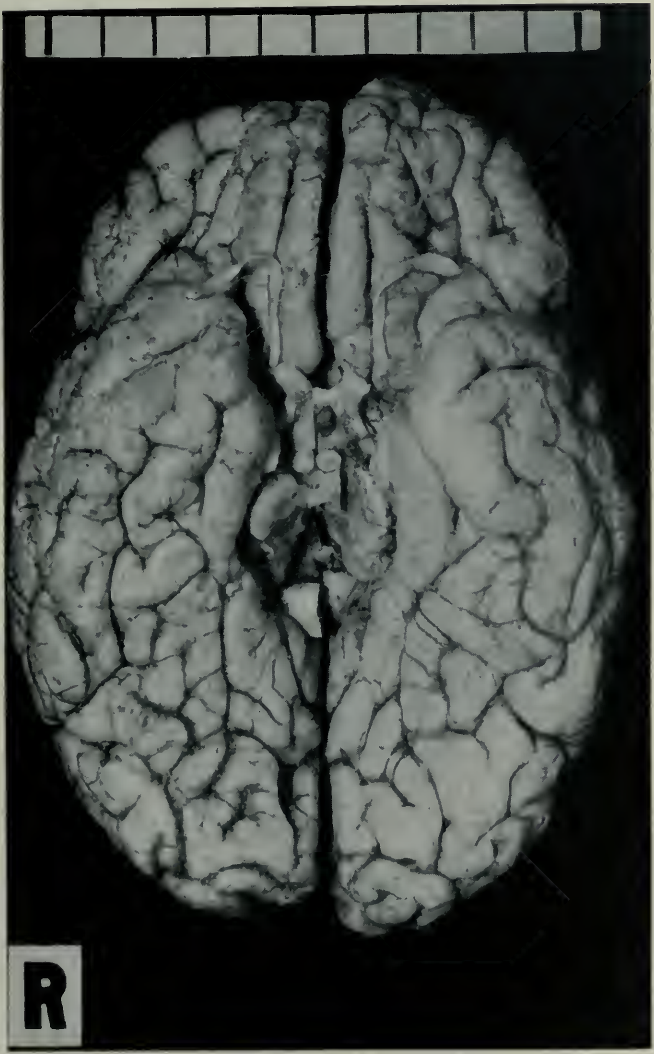

Fig. 2.—Inferior surface of the brain.

Nervous System: General Sensations: She said that she had a feeling of weariness and exhaustion and wanted to sleep all the time. When she arose she was extremely dizzy and sometimes fell.

Eyes: The eyelids were so puffy that her eyes were almost invisible. She complained that her vision was poor; the pupils were unequal, the right being smaller, and they were very sluggish in responding to light.

Ears: She was totally deaf in the right ear, and could only hear when one shouted in the left ear.

Taste and Smell: These were apparently normal.

Cutaneous Sensibility: Tactile appreciation was somewhat impaired. She found it difficult to differentiate between pin prick and pencil. She was not always able to localize for time and space. She complained of numbness of the left arm and leg.

Deep Sensibility: There was no tenderness of the nerve trunk on pressure. The stereognostic sense was good.

Vasomotor Condition: There was an almost general edema, more pronounced in the face, hands and feet, and considerable abdominal ascites.

Reflexes: The knee jerks were sluggish, especially in the left knee. There was no ankle clonus. The organic reflexes were impaired.

Motor Functions: Motility of the facial muscles was poor, owing to the general edema. The tongue protruded in the median line. There was no tremor. The gait was unsteady. Coordination of movements was poor. She was unable to balance herself without assistance, and could not stand in the Romberg position. She had fallen several times, and said this was due to dizziness. She slept all night and the greater part of the day.

Mental Examination.—General Appearance and Attitude: Her face was so waxy and expressionless that with a little "touching up" her resemblance to a clown would be striking. She lay in bed in a sort of stupor most of the time.

Speech: Speech was thick, and she talked in a harsh, raucous voice.

Consciousness and Orientation: She was partially oriented. She knew that she was at Danvers, but was not oriented for time and person. She said it was 1993.

School Knowledge: This was very meager.

Calculation Ability: Her ability to calculate was very poor.

Hallucinations: She had had no hallucinations so far as could be ascertained, and there was no history of them.

Memory: Her memory for remote events was fairly good, but poor for recent events.

Judgment: It was impossible to elicit any definite delusions. She said she did not get along very well with her husband, but denied that it was her fault. She said that everybody tried to boss her around, and she could not stand it any longer. She admitted that she was a little forgetful, but she could remember what happened twenty years ago as well as any one. She was lacking in insight.

Emotions: She was not at all emotional; in fact, she was dull and apathetic. She showed no apprehensiveness, and had not been irritable since she came to the hospital.

Social Relations: She was untidy in her habits, but otherwise caused very little trouble.

Course of the Disease.—March 27, 1919: Slept most of the time. Rather stupid; inordinate appetite.

April 4: She was stupid and sleepy; very deaf; disoriented; no delusions.

April 22: She failed gradually and died today.

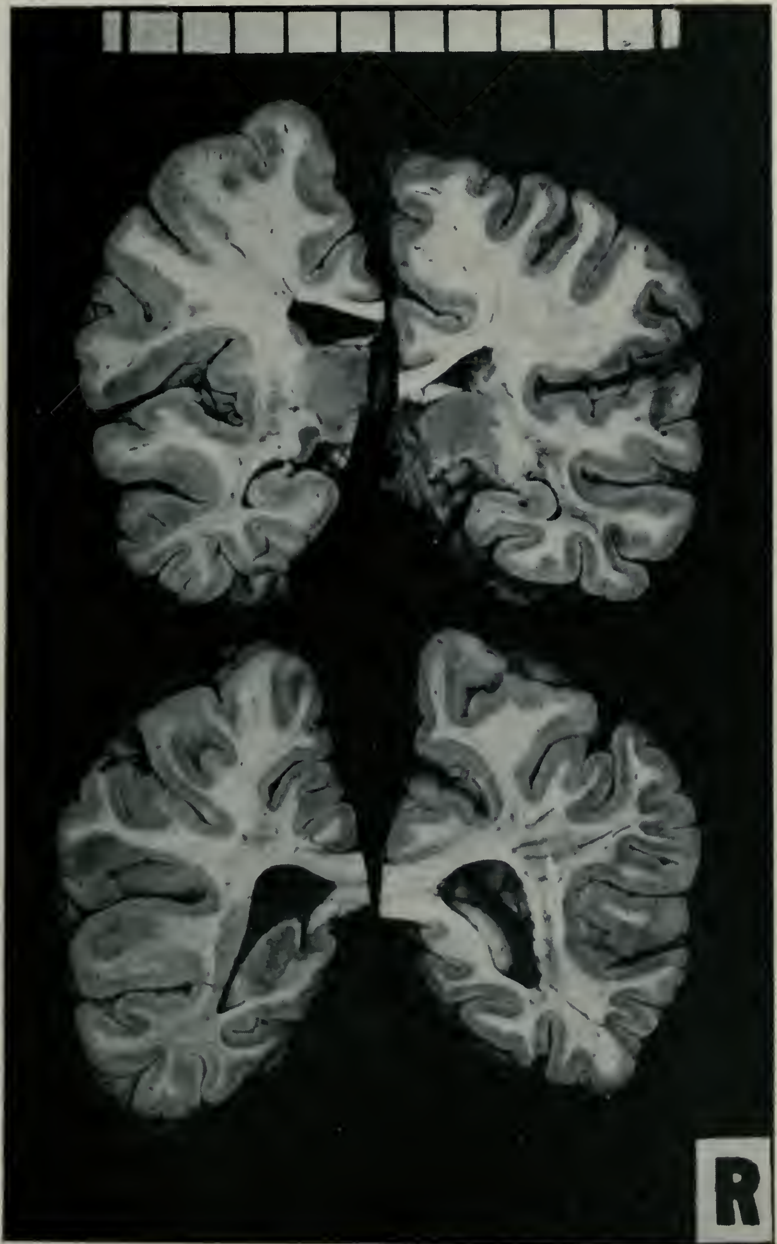

Fig. 3.—Cut surfaces of the brain.

POSTMORTEM EXAMINATION

(Made twenty hours after death)

1. *Gross Anatomy.*—General Description: The body weighed 57 kg.; its length was 150 cm. The body was that of a well developed and well nourished female, aged 66, but apparently older. The skin over the face and neck was yellowish; other parts were grayish white. The face, chest, upper and lower extremities showed edema. There was rigor mortis. The hair was grayish and very sparse, and the head was bald over the vertex. The face was waxy and reminded one of a clown's face. The eyelids were swollen and edematous; the palpebral and orbital conjunctivae were edematous and injected; the pupils were slightly irregular; both measured 4 mm. The nose was broad and swollen, but otherwise not remarkable. The teeth were poor; many were missing. The tongue was thick and coated. There was no discharge from the ears. There was a small nevus in the submental region. The thyroid was not palpable. The chest was well formed. The abdomen was flabby and pendulous. There was a scar extending from the umbilicus to the pubic bone. There were marked striae. There was an exostosis over the upper part of the right tibia. The lymph nodes were not palpable.

Ventral Section: The fat over the chest measured 1 cm., over the abdomen, 2.6 cm., and was very pale in color and of fluid-like consistency. The muscles were pale in color, and were also edematous. The omentum was folded back over the transverse colon, and was rich in fat. The stomach was markedly distended. The appendix was removed; also the right ovary. The uterus was in midline. There were no adhesions around the gallbladder. The spleen was free.

Thoracic Cavity: The mammary vessels stood open on section. The lungs did not meet and were retracted. There were no adhesions around the lungs. The pleural cavities contained free fluid. The pericardial sac was enormously distended, and contained an increased amount of straw-colored fluid.

Heart: It weighed 470 gm. and measured 12 by 13 by 6 cm. The descending branch of the left coronary artery was remarkably sclerosed, and a part of it practically occluded by yellowish atheromatous softening. The measurements were: tricuspid valve, 11.5 cm.; pulmonary valve, 8 cm.; mitral valve, 9.5 cm. The mitral valves were calcified and contracted. The ostium admitted more than three fingers. The aortic valve measured 7 cm. The cusps were thickened. Origins of the coronaries were remarkably calcified. The left ventricle wall measured 2.2 cm., the right ventricle wall, 0.6 cm. The cardiac muscle was pale and soft.

Left Lung: The left lung weighed 400 gm. and measured 22 by 16 by 4 cm. There was no apical scar. The anterior edge of the upper lobe was emphysematous. The surface of the lower lobe was grayish in color. The cut surface was dark red; dark colored blood oozed from the lung; there was no frothy fluid. Sections floated. The bronchial wall was injected and covered by hemorrhagic fibrinous exudation. The peribronchial lymph nodes were enlarged and pigmented.

Right Lung: It weighed 480 gm. and measured 21 by 18 by 5 cm. The anterior border of the middle lobe was emphysematous. There were hardened areas in the lowest lobe. The cut surface showed an irregular, slightly elevated induration around the bronchi. The bronchial wall was markedly injected. On pressure, bloody, frothy fluid oozed from the lung. Sections floated.

Liver: This organ weighed 1,430 gm., and measured 24 by 19 by 7 cm. The capsule was thickened and slightly opaque. The surface was slightly granular. The color was partly greenish blue and partly reddish. The cut surface showed slight evidence of congestion and fatty change. The gallbladder contained small, sandy stones which were dark in color and crumbled easily. The wall of the gallbladder was injected.

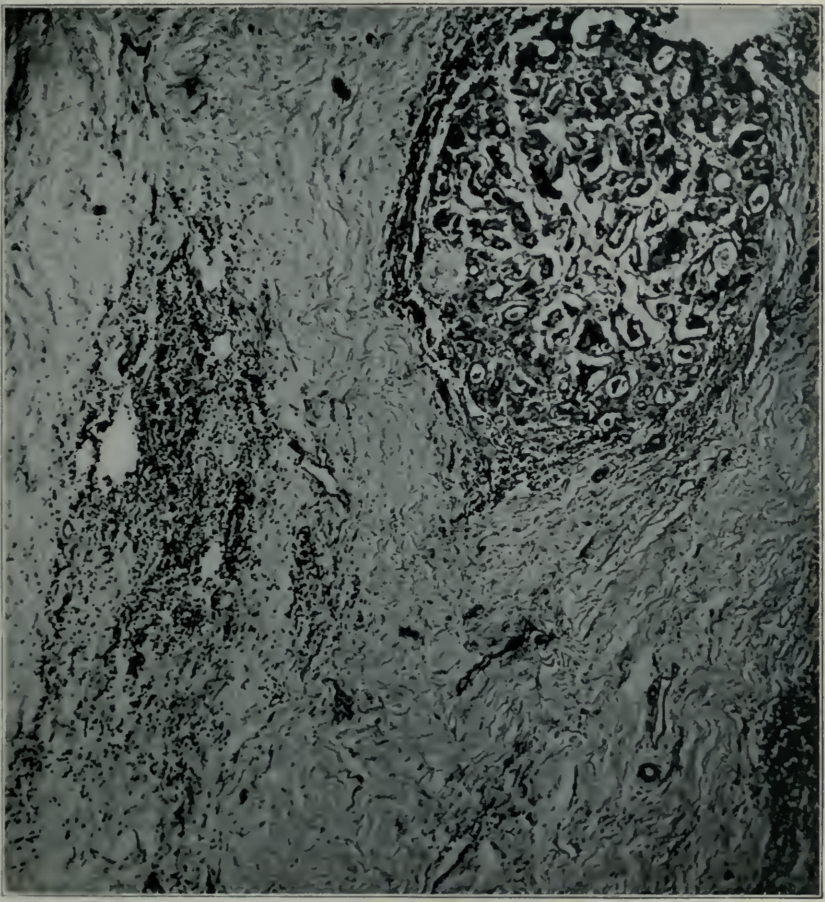

Fig. 4.—The thyroid gland, showing fibrosis and lymphatic cell infiltration. Hematoxylin-eosin staining.

Spleen: It weighed 170 gm. and measured 12 by 8 by 4 cm. It was pear shaped. The capsule was thickened. The surface was granular and clouded. The organ was of unusually hard consistency and there were four fetal lobulations. The cut surface was dark red. The pulp did not bulge and was not easily scraped away. The malpighian bodies were hardly visible.

Left Kidney: This kidney weighed 110 gm. and measured 9.5 by 5 by 3.5 cm. The fatty capsule was increased. The fibrous capsule stripped with some difficulty. The surface of the organ was markedly granular. There were many small cortical cysts. The cut surface was pale in color. The cortex measured 0.4 cm. The pyramids were not well defined and there were whitish striations in the pyramids which suggested increased interstitial tissue. The pelvic fat was remarkably increased.

Right Kidney: The right kidney weighed 110 gm. and measured 10 by 6.5 by 3 cm., otherwise the description was the same as for the other kidney.

Suprarenals: The cut surface showed some increased yellow deposits.

Thyroid: The thyroid was very small; it weighed 10 gm. Careful dissection revealed no isthmus. The cut surface showed no proper structure of the thyroid gland. It appeared to be represented by some fatty and more fibrous tissue. It was fairly firm in consistency.

Parathyroid: The parathyroid glands could not be identified.

Genito-Urinary Tract: The bladder was contracted and contained no fluid. The mucous surface was not remarkable. The vagina was smooth. There were small hemorrhagic areas. The uterus contained turbid fluid; the wall was hemorrhagic and firm in consistency. Section showed abundance of vessels and increase of fibrous tissue. The right ovary was absent, and the broad ligament was adherent to the stump of removed appendix; the left ovary was smaller than normal. The surface was greatly roughened and firm in consistency. Cut surface showed dilated vessels and small cysts.

Gastro-Intestinal Tract: The stomach was distended and contained a greenish mucoid fluid. Mucous membrane was injected. The intestines contained a chocolate colored fluid. The intestinal walls showed a bluish green discoloration, probably of postmortem change. The colon and rectum were not remarkable.

Pancreas: Nothing remarkable was found in the pancreas.

Head: The scalp was thick and adherent to a dense calvarium. There was no diploe. The calvarium was somewhat thickened. The grooves for the middle meningeal arteries were not deep.

Brain: The brain weighed 1,000 gm. The dura mater was thickened. The pia mater was irregularly thickened and showed milky cloudiness over the vertex. The brain was soft and edematous. Superior surface of the brain: There was congestion of the veins, especially on the left side. The pia mater was adherent everywhere over the lateral surface of both hemispheres. A large part of the right posterior central convolution was remarkably narrow. Convolutions of the right cuneus, especially bordering on the margin of the hemisphere, appear much atrophied. The posterior part of the right lingular gyrus was markedly sunken from the surface. The upper part of the left posterior central convolution and left superior parietal convolution was much narrower than normal. The angular gyrus of the left side was also atrophic. Base of the brain: The cranial nerves were not remarkable. The basilar artery and internal carotids were remarkably sclerotic. The pons appeared smaller than normal and resistant to the touch.

Cut surfaces of the brain (Meynert's section): Nothing remarkable was found, except that the above mentioned atrophic convolutions appeared also atrophic on section.

Pituitary Body: This was slightly smaller than normal and was firm in consistency.

Internal Ears: Nothing of note was found in regard to the internal ears.

Spinal Cord: Nothing remarkable was found in the spinal cord.

Anatomic Diagnosis: The body was well developed and well nourished, and there was myxedema of the entire body. The fingers were cyanosed. The conditions noted included alopecia, a waxy clown-like face, irregular pupils, poor teeth, thick tongue, operation scar on the abdomen, exostosis over the upper part of the right tibia, myxedematous condition of the muscle and fatty tissues, ascites, the appendix and the right ovary missing (by operation). There were also hydrothorax, hydropericardium, dilatation and hypertrophy of the heart, arteriosclerosis of the aorta, major arteries of the body and coronaries; also basilar and cerebral arteriosclerosis; increased pericardial fat, emphysema of the lungs, hemorrhagic fibrinous bronchitis, bronchopneumonia of the right lower lobe, congestion and fatty change of the liver, cholelithiasis, a large spleen with increased trabeculation, fatty and fibrous replacement of the thyroid, chronic endometritis and eburnation of the calvarium. Diploe was absent. The brain was small, weighing 1,000 gm. There were chronic pachymeningitis, chronic leptomeningitis and irregular atrophy of the convolutions.

Fig. 5.—Cell sclerosis with fatty degeneration, cells of third layer, superior frontal convolution. Thionin staining.

2. *Microscopic Anatomy.*—(*a*) General Histologic: Thyroid: This was almost completely replaced by fibrous tissue. The ordinary tissue was extremely atrophied, appearing here and there like tiny islands. The thyroid contained practically no colloid matter. The remaining gland tissue was infiltrated and surrounded by small spheroidal or lymphatic cells. In some parts the small lymphatic cells occupied the place of the gland tissue and gave the appearance of the normal lymphatic tissue of the lymph nodes. The small arteries, both in the newly formed lymphatic tissue and in the connective tissue, were remarkably thickened, but showed no perivascular infiltration.

Pituitary Body: The anterior lobe was somewhat smaller than normal and the posterior lobe was remarkably small. The cells of the anterior lobe appeared rather numerous and compact, and the acidophilic cells had increased in the central part as well as in the periphery. The intermediate part extended further into the posterior lobe, but showed no evidence of secretion of colloidal matter. There was slight thickening of the walls of the vessels, and they were infiltrated by lymphocytes, differing in this respect from the vessel picture in the thyroid.

Uterine Wall: There was a formation of a thick layer of newly formed, very vascular tissue over the surface of the mucous membrane.

Ovary: There was a formation of dense new interstitial connective tissue around the walls of the arteries and the surface of the organs. The veins were dilated. Here and there obliterating endarteritis appeared.

Adrenals: These showed a moderate grade of fatty degeneration.

Pancreas: Nothing remarkable was found in the pancreas.

Spleen: Chronic indurative splenitis with abnormally increased pigmentation (hemosiderin) in the cells of the pulp was found.

Lung: The right lower lobe showed exudative pneumonia about the bronchial tubes forming the lobular areas.

Heart: There was brown atrophy of the cardiac muscle with areas of scar formation.

Liver: Chronic congestion and fatty change were found.

(*b*) Microscopical Findings of the Brain: Pieces were taken from the superior frontal, anterior and posterior central, Heschl's transverse and superior parietal convolutions of both hemispheres; also calcarine region, cornu ammonis and several other parts appearing atrophic by macroscopic observation. These pieces were cut by frozen section and by embedding in paraffin, the latter for the purpose of staining by thionin, hematoxylin-eosin, Van Gieson and glia method, the former for fat staining, modified Weigert-Pal's staining and Bielschowsky's silver impregnation.

Hematoxylin-Eosin and Van Gieson's Method: The pia mater was irregularly thickened, especially over parts of convolutions which appeared atrophic. The vessels of the pia mater and upper cortex layer showed marked arteriosclerotic alterations. The thickened parts of the pia mater and the cortex underneath, which often had sunk from the surface, contained numerous amyloid corpuscles. Here and there walls of vessels were calcified. Small vessels appeared to be much increased, disturbing the cell order of the cortex. The width of the cortex was irregular. In highly atrophic parts, such as those mentioned above, and in the neighborhood of the thickened vessels coming down from pia mater, the ganglion cells of the cortex had entirely disappeared, glia cells and glia fibers occupying such areas. The vessels in the deeper layer of the cortex and in the white matter were also thickened and showed marked perivascular gliosis.

Thionin Staining: There was marked disorder of the cells in the superficial cell layers, especially in those of the frontal, anterior central and posterior central convolutions of both sides, right cuneus, posterior part of the right lingular convolution and left angular gyrus. In the deeper parts of the cortex the cell order was fairly regular, although there was some deviation of apical dendrites and some slight disarrangement of the cells. This condition suggested that the cortex layers supplied by short pial vessels had suffered more than the deeper cell layers which were nourished by medullary branches.

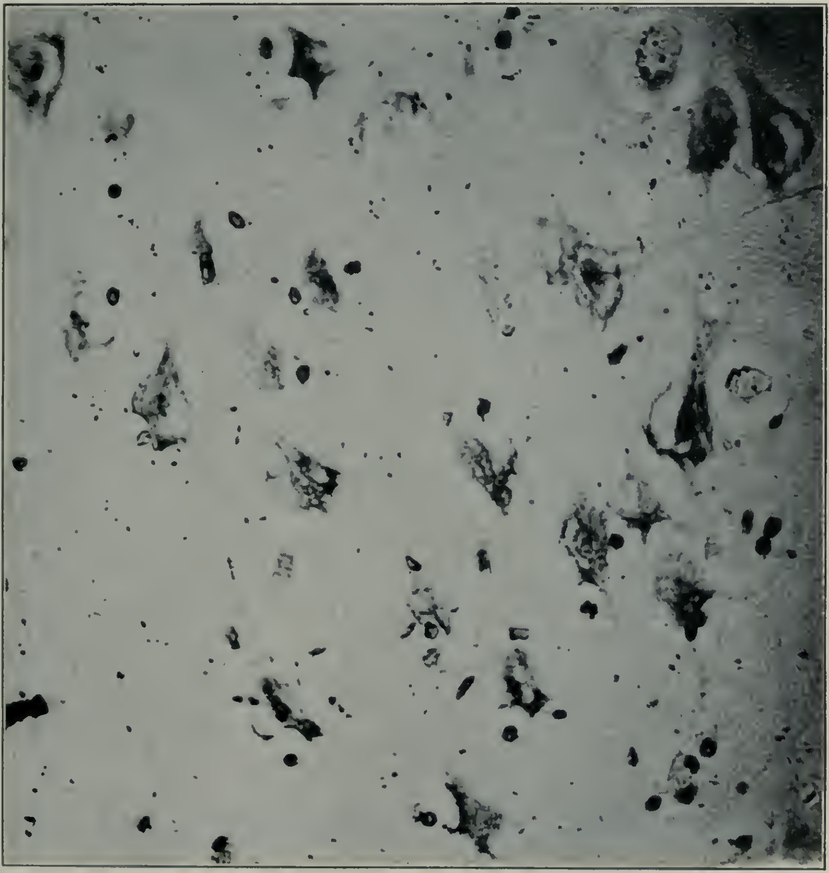

Fig. 6.—Ganglion cells of cornu ammonis showing cell change associated with edematous condition of the brain. Thionin staining.

The cell changes can be divided roughly into three different groups:

1. *Cell Sclerosis* (*Nissl*[4]) *Combined with Fatty Degeneration* (*Fig. 5*).—This change was found in almost all of the cells of the second and third layers

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[4] Nissl: Der gegenwärtige staud du Nervenzellenanatomie und Pathologie, Allg. Ztschr. f. Psychiat. **51**:981, 1894-1895.

(Broadmann) and the majority of the deeper layers. These sclerotic cells showed dark stained nuclei often of elongated, pyramidal or polygonal shape. The protoplasmic body was also darkly stained and remarkably shrunken, making the border between protoplasm and nucleus obscure. The apical dendrites were well stained and traceable to a greater distance than in normal cells. Most of the apical dendrites of the second and third layers took a winding course. These sclerotic cells carried on their side or on their base protoplasmic reticulum which contained greenish and yellowish looking lipoid masses. A few of the apical dendrites contained also a lypoid mass and appeared to be swollen. This fatty sclerotic degeneration was found more markedly in atrophic parts of the cortex than in other places.

2. *Fatty Degeneration with Granular or Heavy Degeneration.*—The cells of the deeper layers (Figs. 5 and 6) and the large cells of the third layer contained a remarkably large amount of fatty pigment. The protoplasmic reticulum containing fatty pigment was located either on the base, the side of the cell or over the apex. The nuclei of these cells were stained a little deeper than normal showing a wheel-like structure or a nebulous, less distinct structure around the nucleus. The nucleus was located eccentrically, owing to the pressure of the deposited fatty substance. It was irregular in most of the cells. The membrane of the nucleus was more or less preserved. Nissl's granules in these cells were either in good condition, finely granulated or had disappeared. Betz' cells, the large cells of the post central region, superior temporal convolutions and calcarine region showed a fairly good condition of Nissl's bodies. In the cells of the other parts granular degeneration predominated or the cells showed dark stained protoplasm with a net-like structure in it.

3. *Cell Change Associated with the Edematous Condition of the Brain (Fig. 6).*—This kind of cell change is found both in the superficial and in the deeper layers, combined with other cell degenerations. Nissl is the first author who gave attention to this condition of the cells. The protoplasmic part was torn apart from the nucleus or from the protoplasm around the nucleus. In some cells a crevice was observed across the width of the protoplasm or a vacuole-like cavity in several parts of the cell, some in the base or sides, others in the apex or even in the apical prolongation. The cavities were irregular in shape and appeared to be made by pulling apart a portion of the cell. At first these cavities were thought to be the remains of fat globules which had been dissolved by alcohol, but it was found that the same cavities were present in formaldehyde fixed specimens stained with scarlet red without the use of fat solvents. In the cornu ammonis the same condition of cavity formation was found around the nucleus showing a wide space between the protoplasm and nucleus. This kind of cell change is said to occur in cases in which the brain is found to be soft and edematous at necropsy. It is possible to cause this kind of cell change by the use of the reagents which withdraw water instantly from the brain tissue. Simchowicz[5] says: "These cell changes are artefact to a certain extent, inasmuch as they are caused by fixing, but they may also be considered pathological, as their formation under fixation is the result of a definite pathologic condition, i. e., an edema of the brain."

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[5] Simchowicz: Histologische Studien über die senile Demenz, Histologische und histopathologische Arbeiten über de Grosshirninde, Nissl, 1911.

In our cases the brain was taken out twenty hours postmortem. Before fixing in formaldehyde it was soft and edematous. We could not, of course, be sure how great a rôle the postmortem change played in this condition. The body, however, was kept cold (25-30 F.) and the weather at the time was very cool, and other brains coming to necropsy under the same conditions showed no such change. Moreover, the skin, fatty tissue and the muscles showed edematous condition due to the disease. There is no reason, therefore, why the edematous condition of the brain should be exempted from the pathologic change due to the myxedematous condition. Admitting that this cell change is partly artefact, caused by fixing solution, it must still be regarded as pathologic because of the body disease and the condition of the brain, which gives rise to this change.

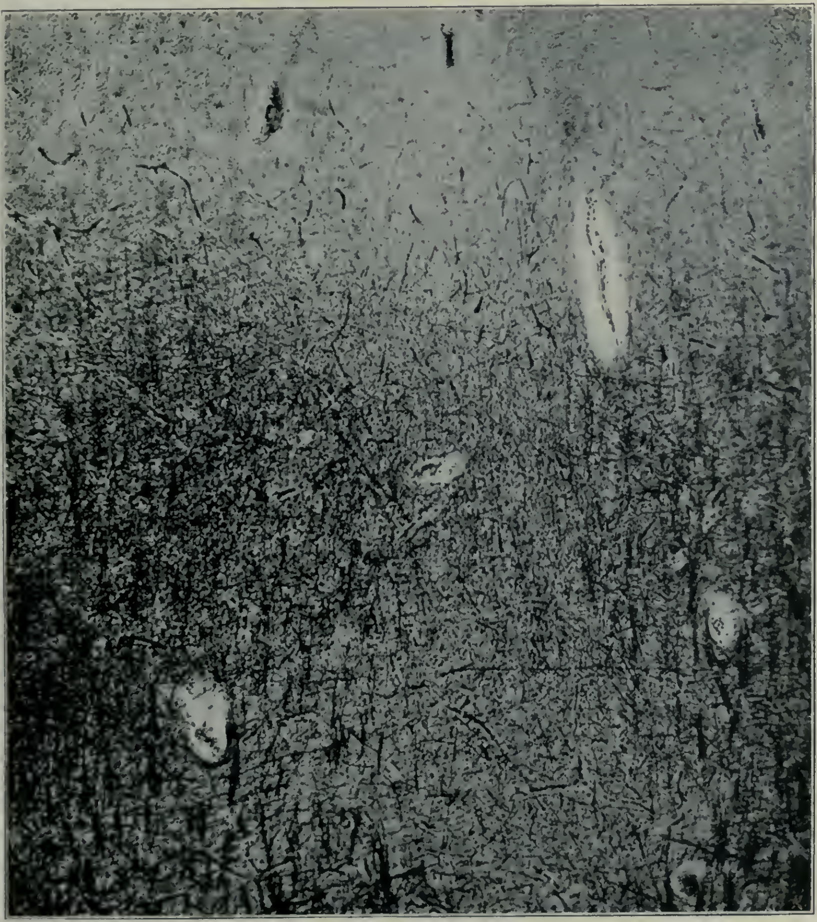

Fig. 7.—Degeneration of tangential fibers and supraradiate cross striations. Weigert-Pal myelin sheath staining.

Bielschowsky's Staining: This staining showed very plainly the alterations of small vessels, especially the so-called arterio-fibrosis of Friedmann, which Simchowicz and Fuller[6] have illustrated in their papers and called attention to as one of the essential findings of senile changes. The arteriofibrosis was found more markedly in vessels of the upper cortex layers. Degeneration of the cortex beneath the pial covering and the scar formation around the arteriosclerotic vessels were observed. Sclerotic cells with fatty degeneration as compared with Nissl's staining showed dark stained protoplasm and nuclei with elongated apical dendrites. Intracellular and extracellular neurofibrils showed segmentation. Larger cells of the deeper cortex layer possessed pale stained nuclei and fairly well preserved intracellular neurofibrils. Fatty degeneration and cell changes caused by the edematous condition of the brain tissue were also observed.

Scarlet Red Staining: Fairly abundant fat corpuscle cells were found in thickened parts of the pia mater. The walls of the vessels of the pia mater and of the brain showed fatty degeneration. Fat corpuscle cells were found around the vessels and the highly degenerated cortex parts. Fatty degeneration of the ganglion cells is one of the most striking features of this case. All cells of the upper and the deeper cortex layers showed a deposit of an extremely large amount of fatty pigment substance. This condition existed over the entire brain tissue, while in the degenerated cortex and in the cornu ammonis it was perhaps most prominent. Spider cells of the surface layer and the large pale stained nucleus of the glia cells showed marked fatty degeneration.

Myelin Sheath Staining (Fig. 7): Tangential fibers were to a marked degree destroyed, here and there showing the remains of fine fibers and some hypertrophic ones. In the highly degenerated cortex the myelin sheaths are lost. Supraradiate cross striations (Supraradiaere Flechtwerke) were scarce. In the white matter there were degenerations running longitudinally with the vessels.

Glia: The glia cells were increased, especially in the deeper parts of the cortex and the white matter. Small dark stained nuclei were not so numerous as pale stained, larger forms with large nuclei which were often stained metachromatically. Around these were often observed (by thionin staining) yellowish and greenish appearing lipoid granules which stained markedly with sudan or scarlet red. This condition of lipoid accumulation around the nuclei was seen more distinctly in the cortex than in the white matter. In the neighborhood of the vessels of the white matter and the cortex, glia cells were remarkably increased.

Weigert's Glia Staining (Fig. 8): The surface glia layer was distinctly broader than normal, some of the fibers reaching deep into the cortex. The glia fibers were also increased remarkably in the atrophic parts of the cortex and around the thickened vessels. On the cortex surface where the atrophic parts had sunk from the normal level, the glia fibers were denser and penetrated deeper into the cortex. The whole feature of this picture of increased glia fibers is like a wedge form. The glia fibers consisted of a small number of thick fibers and a large number of finer and delicate ones. There were also areas in which glia fibers were growing into the pia mater, deep into the pial tissue. Corpora amylacea were very abundant in the atrophic parts of the cortex and the pia mater, where the glia fiber proliferation was most prominent.

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[6] Fuller: Further Observations on Alzheimer's Disease, Proceedings of the American Medico-Psychological Association, Sixty-Eighth Annual Meeting, 1912.

(*c*) Microscopic Examination of the Cerebellum: Scarlet Red Staining for Fatty Degeneration: Purkinje cells stained diffuse brownish, showed no fat corpuscles or granules. Dendrites were not visible. There was a small number of fat corpuscle cells in the Purkinje cell layer. Walls of capillaries and arteries showed fatty degeneration and a small number of fat corpuscle cells were seen around them. There were a few corpora amylacea in the deeper part of the molecular layer and Purkinje cell layer.

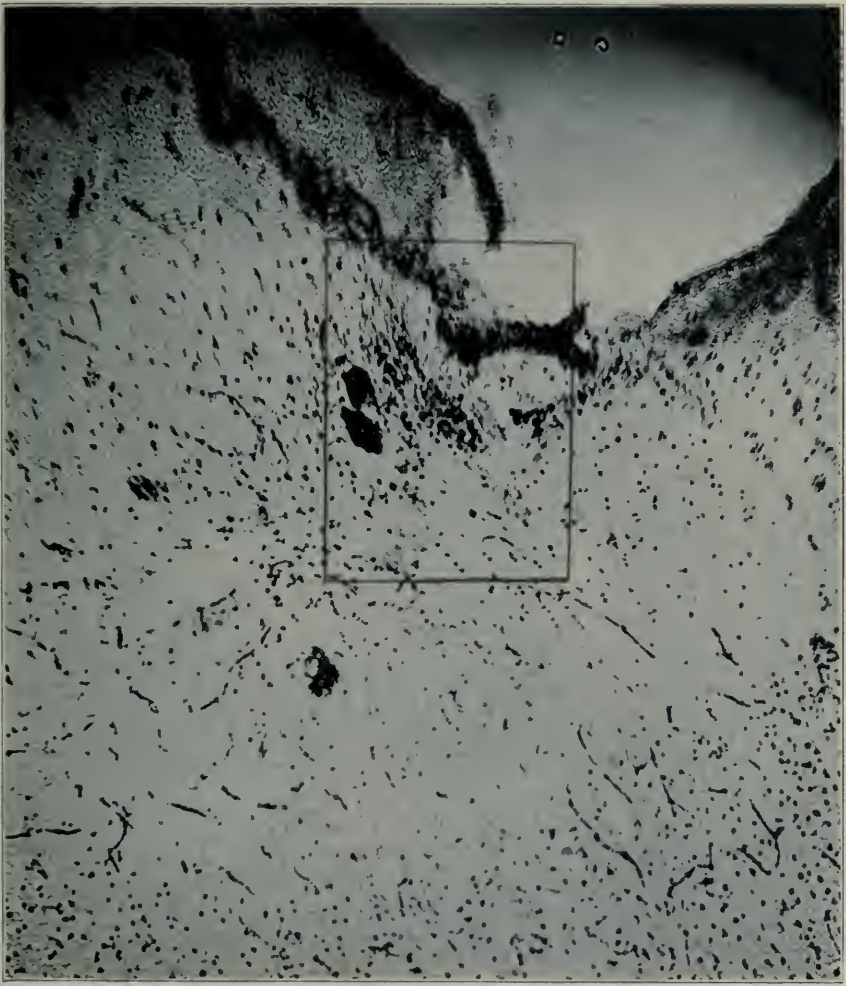

Fig. 8.—Thickening of the surface glia layer, thickening of the walls of vessels, luxuriant growth of the glia fibers and accumulation of amyloid corpuscles. Weigert's glia staining.

Bielschowsky's Staining for Neurofibrils (Fig. 5): The molecular layer was reduced in width. The cells in this layer, both those in the upper part and those in the deeper zone, were remarkably decreased. Tangential fibers showed also diminution and they took a wavy course. The Purkinje cells were markedly destroyed, and those remaining appeared smaller and poorly stained, though an epicellular and endocellular network existed. Dendrites were for the most part absent, so that the molecular layer appeared much lighter; basket fibers and cushion fibers were diminished. The granular layer also appeared paler on account of the scarcity of the small spheroidal ganglion cells. Remarkably swollen fibers, whose origin was hardly determined, were found both in the molecular and granular layers. They might be either basket fibers, afferent fibers or nerve prolongations of Cajal's cells of the granular layer.

Some of the axis cylinders were swollen (not far from the Purkinje cells) to a peculiar shape, spindle, spheric, sac form or other shape. Neurofibrils in this swollen body appeared mostly homogeneous, taking diffusely silver stain. This latter finding is the same as that described and explained first by Cajal and later by Rossi, Marinesco[7] and Schaffer.[8] These authors found this peculiar local swelling of axis cylinders in some special diseases, and claimed that they were the result of a regenerative process of the axis cylinder, the continuity of which is disturbed. I have seen this peculiar swelling of the axis cylinder in several kinds of brain disease, and I am rather of the opinion that this is a kind of regenerative-degenerative process, seen in many kinds of diseases in which there is degeneration of the Purkinje cells. Accurate description and explanation of this change is not the purpose of this paper, but will be given in a later communication. The vessels appeared to be very much increased in number in all the cortex layers and in the white matter of the cerebellum. Each capillary showed marked arteriofibrosis, probably more marked than in the cerebrum.

(*d*) Microscopic Examination of the Spinal Cord: Little of note was found with the exception of a slight degree of arteriosclerosis and fatty degeneration of the cells of the anterior horn.

THE AUTHOR'S CASE COMPARED WITH CASES IN LITERATURE

The onset of the myxedema in this case is unknown. It appears to have begun ten years ago when the patient was operated on for broad ligament cysts. Since that time the patient had been slowly but progressively failing, both mentally and physically. Her memory had begun to fail, especially for recent events. She had had many attacks of vertigo, sometimes falling to the floor, and this condition had become much more conspicuous in the later stages of the disease; she could not stand in the Romberg position, her gait became unsteady, and she showed marked disturbances of coordination movements. The latter symptoms remind one of the cerebellar symptoms of the myxedematous patient, which has been reported by Soderbergh, though accurate examination for these symptoms was not made in the present case. In recent years she had become untidy and would wander about on the streets. At times she became depressed and threatened suicide.

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[7] Marinesco: Nouvelles contributions à l'étude de la régénérescence des fibres du système nerveux central, Jour. f. Neurol. u. Psychol. No. 17, 116, 1910.

[8] Schaffer: Zum normalen und pathologischen Fibrillenbau der Kleinhirnrinde, Allg. Ztschr. f. d. ges Neurol. u. Psychiat. **21**:1, 1914.

Cases like this have also been described by Pilcz and others. Later, before and after her admission to the hospital, she became very dull and stupid, sleeping all the time.

The physical symptoms were typical. There was edema over the whole body. Bald head, "clown-like" face, thick flabby tongue, poor condition of the teeth, deafness, hydrops of the serous cavities, cyanosis of the hands, diminished activity of the reflexes, arteriosclerosis, bradycardia, etc., all correspond to the typical case.

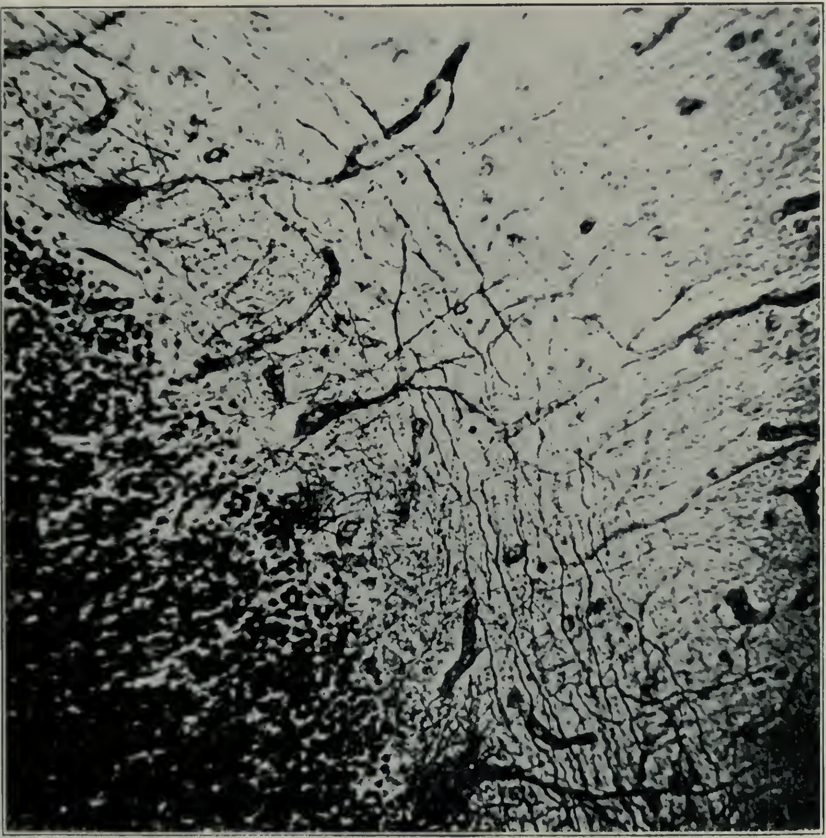

Fig. 9.—Swelling of the axis cylinder of the Purkinje cell (middle), emaciated cell body of the same cell. Bielschowsky silver impregnation method.

Gross anatomy showed an extremely atrophic thyroid gland, both sides equally small, and with no isthmus. In our first case in the Danvers State Hospital series, according to the description of Dr. Peabody, the thyroid consisted of two small, tough, fibrous masses not connected by any isthmus, lying one on either side of the cricoid cartilage and beginning trachea. On section it looked like pale fatty tissue with much connective tissue. The parathyroid in that case, as well as in the present case, could not be identified. Although the absence of the isthmus is not uncommon, it certainly is not typical. It makes me doubt, therefore, whether there is any relationship between congenital malformation and this disease.

Necropsy revealed the oöphorectomy of the right side and the appendicectomy. It is very interesting to have found the same condition in our former case about which the necropsy protocol says: "Appendix is absent. No tube and ovary on the right. The left tube is invisible in the mass of adhesions. The left ovary is small (residual), and there are thick fibrous adhesions between the ovary and the adjoining tissues and to the ileum." This fact, found both in our former and present cases, suggests to me a probable etiologic relationship between the pathologic condition of ovaries and the myxedema.

Microscopic examination revealed extreme atrophy of the thyroid gland replaced by fibrous tissue and infiltrated by lymphatic cells. The pituitary body was a little smaller than normal, contrary to the statements of authors such as Boyce and Beadles, Ponfick and Murray, who found enlargement of the pituitary body in this disease. Arteriosclerosis of remarkable degree was found in all vessels of the body and the brain. In our former case there was also marked arteriosclerosis throughout the aorta.

As for the brain, cerebellum and spinal cord, we found more or less remarkable pathologic changes differing from the description of some authors such as Hun, Prudden, West and Harely. But are these pathologic findings specific or characteristic of this disease? This is an important question.

Before answering this question, let us survey the nature of these pathologic changes. They can be divided into two groups, the one being more or less localized and the other general in character. The first group is expressed by localized degeneration in certain convolutions, degenerations around the sclerotic vessels of the superficial cortex layers, perivascular gliosis, perivascular and focal accumulation of fat corpuscle cells, destruction of myelin sheaths, proliferation of glia fibers in degenerated parts, irregular thickening of the pia mater and the surface glia layer, etc. Vessels of the pia mater and the superficial cortex layers showed more marked sclerosis than those in deeper layers. The same condition obtained in the arteriofibrosis of small vessels. This group of changes, however, is nothing but the arteriosclerotic alteration of the brain. The second group consists of universal changes, such as fatty degeneration with granular and heavy change (Nissl), cell sclerosis with fatty degeneration, and the cell vacuoles associated with the edematous condition of the brain. Though the fatty degeneration and cell sclerosis are found in the arteriosclerotic nature of the alteration, they are not universally distributed over the whole of the brain. According to Simchowicz, "Pathologically arteriosclerotic alteration is focal in character, whereas senile changes show diffuse distribution." So the second group of changes is different from those of an arteriosclerotic nature and resembles more the normal senile alteration.

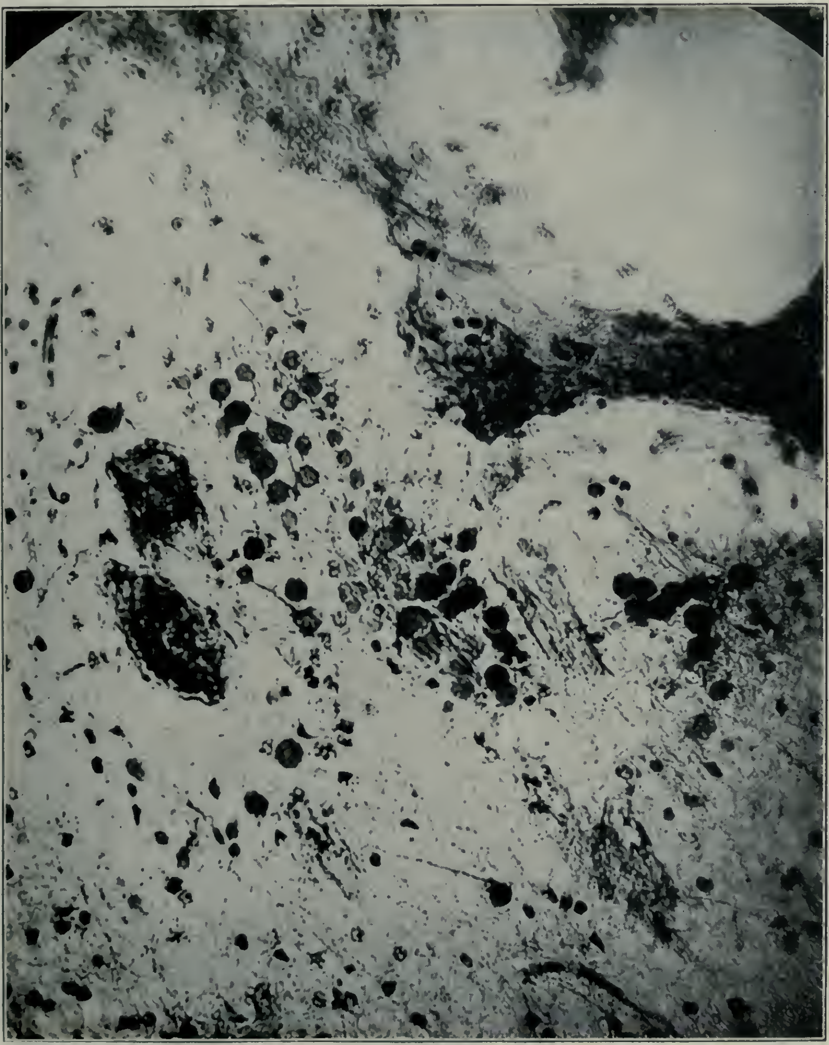

Fig. 10.—Enlarged picture from section of Figure 8.

We have, then, a case showing the combination of arteriosclerosis and senile changes. Now the question is: Are these arteriosclerotic alterations with senile changes due to the myxedema, or are they independent of it? The patient was 66 years old, and it is quite possible to show arteriosclerotic alterations and even senile changes without any disturbance of the internal secretion. But one thing should be mentioned — the fatty degeneration of the ganglion cells is too great for the normal senile changes at this age, and that makes the question much more complicated.

It is, however, a common occurrence in senility for the thyroid to become atrophic and undergo diffuse sclerosis, destroying the gland tissue. Horsley declares that old age is only a form of mitigated hypothyroidism, and that the people who enjoy a green old age owe this happy condition to a thyroid which has remained normal. "The points of resemblance," says Crotti,[9] "between senility and a slight degree of hypothyroidism are more than one. Falling out of the hair, deafness, falling of the teeth, dryness of the skin, arteriosclerosis, bradycardia, diminution of the function of the nervous system and other symptoms which occur both in old people and hypothyroidism, can all be attributed to the atrophic condition of the thyroid gland." If this hypothesis is true, the question is very much simplified. For the increased arteriosclerosis and senile changes are nothing but the manifestation of the diminished function of the thyroid gland, and the pathologic findings which have been observed can safely be attributed to the hypothyroidism.

Schnitzler[10] described a case of a 32-year-old female, who had shown marked clinical symptoms of myxedema and mental symptoms of apathy and stupor and somnolence. He grouped his case with the type of which Spielmeyer wrote as "causes of senile dementia which differ from the usual type in that the marked dementia rapidly ensues, together with focal symptoms of asymbolic and aphasic character." He shows, however, as Fuller expressed it, an inclination to flirt with the rather fascinating idea of an origin from disordered internal secretions, since his case and one of the cases reported by Perusini exhibited certain myxedematous symptoms. "Although," Schnitzler says, "the thyroid gland could not be examined postmortem, since the patient became very much better under the administration of thyroid preparation, it is quite possible that we dealt with a thyrogeneous disease. So we have a case, showing a possibility of an intimate relationship between internal secretion and senile degeneration. If the normal senile degeneration is merely a consequence of hypofunction of the thyroid gland, can this condition of senile changes in our case not be attributed to the complication of the myxedema?" It seems to me rather unnatural to have classed his case as senile dementia. I see no reason why it should not be grouped with the myxedematous psychoses. In his case he has found no senile plaques; he has observed Alzheimer's neurofibrillar change, but this is not at all pathognomonic of senile dementia or of other senile conditions. Goto[11] found typical Alzheimer's[12] change in feebleminded subjects, and even in dementia precox. I was unable to find any of these changes in any parts of the brain in this case.

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[9] Grotti: Thyroid and Thymus, Philadelphia, Lea & Febiger, 1918.

[10] Schnitzler: Zur Abgrenzung der sogenannten Alzheimerschen Krankheit, Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:34, 1911.

Further, if we look over the mental symptoms, such as loss of memory, especially for recent events, disturbance of the attention, suspiciousness, acute dementia, etc., which are always found in senile cases as well as in this case, we cannot believe that the anatomic substrata of this disease is dependent on anything else than the senile changes in a broad sense — i. e., arteriosclerotic alterations, normal senile alterations and characteristic degeneration of senile dementia.

As for the cell change associated with the edematous condition, though it has, of course, nothing to do with senility, it must be considered as pathognomonic, since the edematous condition itself is the partial manifestation of the general symptoms of this disease.

The cerebellum was found more intensively degenerated than the cerebrum. The Purkinje cells diminished in number, those remaining showing marked degeneration. The fatty degeneration of the Purkinje cells, however, was not so marked as of the ganglion cells of the brain and the spinal cord which shows merely that the Purkinje cells are so-called lipophobic cells and nearly always escape fatty degeneration.

How far the cerebellar symptoms of reported cases and of the present case can be attributed to the pathologic anatomic conditions of the cerebellum is hard to answer, but it is quite probable that they have played more or less of a part in the existence of such marked symptoms.

SUMMARY

1. The writer presents a case of typical myxedematous psychosis which showed, together with known symptoms, marked disturbance of coordination, vertigo and somnolence.

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[11] Goto: Alzheimersche Neurofibrillenveraenderungen in der Hirnrinde der Schwachsinnigen, Tokio Shinkeigakkai Zasshi, 1917.

[12] Alzhemer: Die arteriosklerotische Atrophie des Gehirns, Allg. Ztschr. f. Psychiat. **51**:809, 1895. Beitrag zur pathologische Anatomie der Seelenstoerungen des Greisenalters, Neurol. Centralbl. **18**:95, 1899. Seelenstoerungen auf arteriosklerotischer Grundlage, Allg. Ztschr. f. Psychiat **59**:695, 1902.

2. The thyroid gland was found extremely atrophic with the gland tissue replaced by connective tissue fibers and with no isthmus. The remaining gland tissue was infiltrated by lymphatic cells. The writer suspects some etiologic relationship between the congenital factor and this disease.

3. The pituitary body was smaller than normal, though microscopically there was no evidence of atrophy.

4. The right ovary was removed ten years ago, the left remained atrophic, showing an evidence of chronic inflammatory process.

5. The writer suspects, having seen a similar condition of ovaries in a former case of the Danvers State Hospital series, some etiologic factors in certain diseased conditions of the ovaries.

6. The pathologic changes in the brain and the cerebellum consist of arteriosclerotic alterations, general senile changes and cell change associated with the edematous condition.

7. The writer attributes the arteriosclerotic alterations and the general senile changes to the effect of the myxedematous disease.

8. The cell change associated with the edematous condition is considered as pathognomonic.

The writer believes also that there is a possible correlation between the marked atrophy of the cerebellum and the clinical symptoms of disturbed coordination and of vertigo.

The writer is indebted to the staff of the Danvers State Hospital, especially to Drs. Macdonald, Bryan and Stevenson, for completing this report. He is also indebted to Drs. Southard and Canavan for their encouragement and advice in his study. To all of these he wishes to express his appreciation.

In addition to the references already given, the following will be found of interest:

Barham, G. F.: Insanity with Myxedema, J. Ment. Sc. **58**:*226*, 1912.

Alzheimer: Ueber eine eigenartige Erkrankung der Hirnrinde, Allg. Ztschr. f. Psychiat. **64**:146, 1907.

SENSORY CHANGES IN INJURIES OF THE MUSCULOSPIRAL NERVE *

ARTHUR S. HAMILTON, M.D.
MINNEAPOLIS

In view of the several very brilliant and equally exhaustive reports on the innervation of the skin which have appeared in the literature of the last few years, one must, with some temerity, approach the study of the sensory functions of any one of the peripheral nerves. And this is the more true when one considers that much of the work recently published has been done on patients well fitted both by education and by training to carefully analyze their sensations and by workers who have given to the task many months of painstaking examination, whereas the conditions obtaining in army life render a similar study wholly impossible, partly by lack of that preliminary education and training on the part of the patient which is essential to the best results in interpreting new and confusing sensations, and partly by reason of the lack of time which permits only a limited study in a routine examination. The difficulty which the latter factor offers is appreciated when one recalls the statement of Trotter and Davis[1] that it requires from forty-five to fifty hours to examine and record the sensibility to cold alone in a fairly large area. For these reasons, therefore, no attempt has been made to describe adequately and to measure all the different sensory disturbances that may appear in the region supplied by the musculospiral nerve. The excuse for this report must lie in the scarcity of literature dealing with the sensory field of this particular nerve and in its surprising areas of distribution found in numerous cases of nerve wounds among soldiers — surprising at least in view of the anatomic distribution of the nerve as commonly taught.

It is important to mention that, as all the cases here reported were studied at the Walter Reed General Hospital in Washington, D. C., it was not possible to determine the area of sensory change in the early stages of the injury and in several instances well marked evidences of healing had appeared before the study was begun. It was especially in those cases in which the loss of function of the musculospiral nerve was dependent on fracture of the humerus, with the development of scar tissue and which presented a cessation of function but not necessarily an anatomic loss of continuity, that the picture was at times

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* Read before the meeting of the American Neurological Association, held in Atlantic City, N. J., June 16-18, 1919.

[1] Trotter and Davis, Brain **38:**137 (Feb. 9) 1919.

greatly altered, for in from three to five or six months that had usually intervened before the study was made, marked improvement could, and often had occurred.

In many cases the amount of destruction of tissue, in addition to the actual section of the nerve, has seriously interfered with the work, often by the injury or section of nerves, especially of the musculocutaneous, in addition to the one desired to be studied, thereby confusing the sensory picture. So far as possible, however, cases were chosen for special study in which the section of the nerve appeared to have been at least physiologically complete, in which a sufficient period of time for healing to occur had not intervened, in which the wound was small and appeared not to have involved nerves other than the musculospiral and in which the intelligence of the patient was such as to permit a reasonably reliable study. It must be admitted, however, that when all these restrictions were applied, the number of cases suitable for study was so much reduced that the limits were more than once overstepped.

The total number of musculospiral cases studied was fifty-five, and in all of these the investigation was carried far enough to determine the area of sensibility to pressure, pin prick and cotton or camel's hair touch, but beyond these routine tests, the type of study made varied so much and in many of the cases is still so incomplete that no absolute percentages of the difference findings can be given.

THE SENSORY BRANCHES OF THE MUSCULOSPIRAL NERVE

There are three sensory branches of the musculospiral nerve, distributed in the average case as follows:

The internal cutaneous branch is given off in the axillary fossa and supplies the internal part of the posterior surface of the upper arm below the deltoid and as far down as the point of the elbow. On account of its high origin, this branch is affected in only a small number of cases, in which the musculospiral nerve is injured at a very high point. The external cutaneous branch is given off at a point where the musculospiral nerve passes around the external edge of the humerous and supplies the external part of the posterior surface of the arm, the lower part of the outer surface of the arm and a more or less narrow band of the posterior surface of the forearm lying between the musculocutaneous and the internal cutaneous nerves. Often there are two external cutaneous branches, and in such cases the area of numbness will be different if only one branch is involved. The superficial radial nerve has its origin at, or just below, the elbow joint, and after passing down the forearm is distributed to the skin of the external part of the dorsum of the hand, to a variable but usually small part of the thenar eminence, to the dorsal surface of the thumb, to the dorsal surface of the first phalanx of the index finger and to the outer half of the dorsal surface of the first phalanx of the second finger (Fig. 1).

With so wide an anatomic distribution and with its well known extensive motor supply, one might expect a very widespread and complete loss of sensation in the various injuries to which the musculospiral nerve is subjected. As a matter of fact, quite the contrary is true, and the area of sensory loss is sometimes surprisingly small and is almost always incomplete.

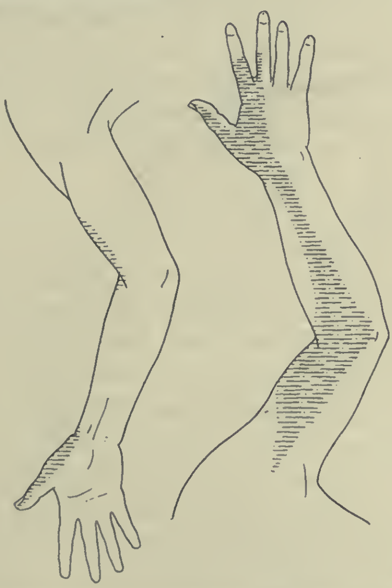

Fig. 1.—Area of sensory distribution of the musculospiral nerve as given by Tinel in Nerve Wounds, p. 103.

One of the unexpected findings in all nerve injuries is the failure of the individual to recognize subjectively and with any degree of accuracy the area of sensory loss, especially of epicritic loss and particularly so in the area of distribution of the musculospiral nerve. Frequently patients with well marked paralysis of the extensors of the hand, following injury, if asked for their subjective sensory area will say they have none. If their attention is insistently brought to the matter, they will tell of a small area of numbness over the first interosseous space and over the dorsum of the thumb and perhaps over the dorsum of the first phalanx of the index finger, and they will be greatly surprised when a larger area of sensory loss can be demonstrated.

As already explained, the internal cutaneous branch rises so high that it is seldom involved in injury of the musculospiral nerve and even the external cutaneous frequently rises above the area of injury, at least its supposed area is often found not involved even in injuries high up in the arm. It is impossible, however, to explain all the variations found in the area of numbness on the basis of the point of origin of the sensory branches, and other explanations are offered, chiefly that of the overlapping or anastomosis of the musculocutaneous, internal cutaneous, and median nerves involving the musculospiral field and giving varying areas of sensory loss when these nerves are injured. In the upper arm the intercostohumeral or the lesser internal cutaneous may supply areas ordinarily innervated by the internal cutaneous branch of the musculospiral and may also supply that part of the upper arm ordinarily supplied by the external cutaneous branch. I have also recently seen a case in which a lesion of the circumflex nerve caused a large area of numbness in that part of the upper arm ordinarily supplied by the external cutaneous branch of the musculospiral nerve.

As is so common after severance of any large nerve of the arm, the patient will usually complain of complete loss of feeling in all parts of the arm distal to the wound, directly following injury to the musculospiral nerve. During this preliminary period the arm is described as a very heavy appendage, or the statement is made that the arm feels as if wholly separated from the body and must be seen or felt by the other hand in order that the patient may appreciate its presence. After the lapse of a few hours or days, the patient discovers that there is feeling in the parts not supplied by the nerve immediately injured and the area really involved is more or less clearly recognized.

In the fifty-five cases that I studied there was only one instance in which the musculospiral nerve alone had been severed sufficiently high to produce a loss of sensation corresponding to all three branches.

REPORT OF CASES

CASE 1.—The previous history was not noteworthy. October 13, 1918, the patient while holding his left arm in the position of guard with the forearm horizontal, was struck below the clavicle by a shell fragment which punctured the lung and passed out the back. His left arm immediately became numb and fell to his side. There was no jerking or twitching. He felt as if the arm were shot off. His neck was stiff and he was unable to look down. He repeatedly asked those about him if his arm were not really gone. For some time after the injury he would seem to feel his arm in the position of guard and would reach up with his other arm to scratch it. At first he had excessive pain in the shoulder; later his whole arm ached a good deal, but he could not localize the ache as any worse in one part than in another. There was a slight causalgia for a time in the median. He was told that his musculospiral nerve was sutured directly after his injury, but the record is not clear on this point. At the examination, in February, 1919, he was conscious only of some numbness on the dorsum of the index finger, which he thought worse in the first phalanx than in the second, and of some numbness on the back of the upper arm. An examination made with considerable difficulty, at this time, showed sensory changes as seen in Figure 2. Since February there has been some improvement in the sensory and motor condition and on May 31 even epicritic sensibility had returned over the dorsum of the hand with the exception of the area over the first metacarpal bone. The analgesic islands, outlined in the diagram, persisted. The dorsum of the first phalanx of the index finger was anesthetic and hypalgesic. Tinel's sign was elicited as far as the elbow and at this point stroking produced a tingling feeling in the fingers.

In a general way this case conforms fairly well to the typical distribution of the musculospiral nerve. The fact that sensibility is more disturbed in the upper part of the forearm than at the wrist was a frequent finding in our cases. The enlargement of the sensory area at the wrist and just above is atypical, though found in one other case, and on stroking touch the patient was able to identify an area of decreased sensibility even to the extreme ulnar side of the arm, just above the wrist joint.

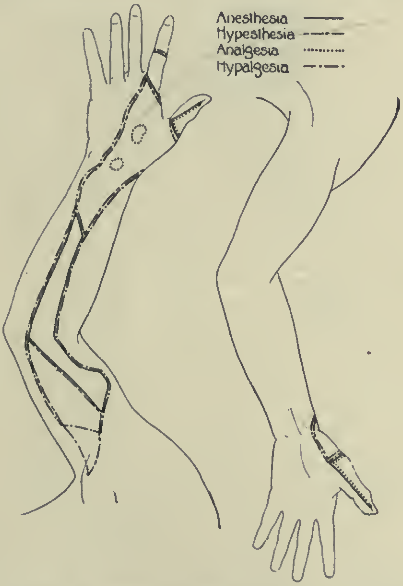

Fig. 2 (Case 1).—Area of sensory disturbance. In this and in the accompanying illustrations the meaning of the legends employed is given as a part of each illustration. When lines lie close together it is not intended that they should represent the relative limitations of the different sensory fields. They were not permitted to overlap merely as a matter of clearness of representation. In most cases also the work was not sufficiently carefully done to permit the accurate tracing of the relative sizes of the different fields.

In the fifty-five cases there were twenty-seven with a definite area of sensory involvement in the forearm as well as the hand, and one instance of involvement of both musculospirals, each case involving the forearm. Seven of these were rejected on account of various complications. The subjective sensory phenomena were usually about as in Case 1, with an initial sensation much as if a heavy blow had struck the arm and a subsequent feeling of loss of the limb and a still later more clearly defined and localized loss of sensibility. The following is a fairly typical case except that there seems to have been less initial loss of sensibility than was ordinarily present.

Case 2.—While standing the patient was struck by a rifle bullet in the upper portion of the left upper arm, Sept. 15, 1918. The arm was in a horizontal position, and he had the sensation of being struck a heavy blow. The arm rotated inward, bringing the palm downward. The fingers flexed like a claw and remained so for some time. There was no pain except at the first blow and no feeling of the arm being gone. He did not recall noticing much numbness at any time. His record states that he was operated on the night of the 15th and his musculospiral nerve sutured.

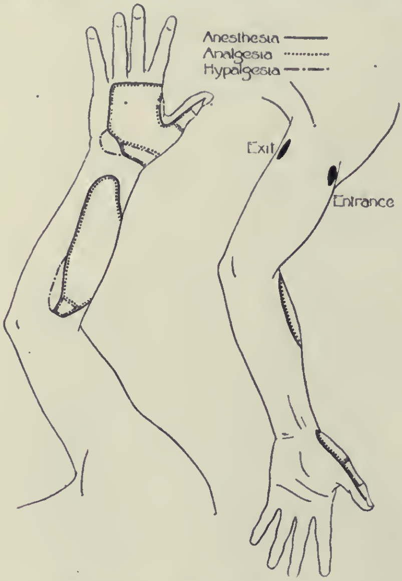

Fig. 3 (Case 2).—Area of sensory disturbance.

When examined, May 7, 1919, a rather extensive area of sensory disturbance was found (Fig. 3). There was no connection between the area on the forearm, which extended well over into the supposed region of the musculocutaneous nerve, as did some of the others, and the area on the hand. There was a loss of sensibility to cold at 2 C. slightly wider than the area of touch loss on the forearm and especially extending considerably farther over on the palmar surface of the forearm than any other form of loss of sensibility. Two point discrimination was greatly decreased on the dorsum of the forearm (33 per cent. of correct answers at 110 mm. with 53 per cent. of correct answers at 50 mm. on the right forearm). On the dorsum of the hand two point sensibility could not be measured on account of the inability to perceive pressure.

Joint sensibility was normal in the fingers and thumb. Vibration sense was normal in the fingers and somewhat reduced over the thumb.

Case 3.—While kneeling and digging in, the patient was struck by shrapnel in two places on the left upper arm and in six or seven places on the right upper arm, Oct. 5, 1918. He was also struck in the right knee and thigh. He fell and was unconscious at least a few minutes. On recovering consciousness, both hands and arms were wholly paralyzed for motion and sensation and remained so for one day. The left hand and forearm felt numb on the dorsum until the end of January and then began rapidly to improve. There was some numbness in the musculospiral area of the right hand and lower part of the forearm for two weeks with rapid improvement following.

When examined, May 9, 1919, there was a slight hypesthesia and hypalgesia in the right hand with a subjective area practically the same in extent (Fig. 4).

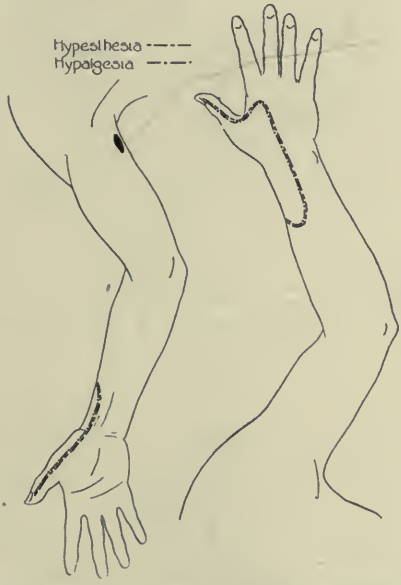

Fig. 4 (Case 3).—Area of sensory disturbance.

May 28, 1919, it was impossible to map any definite area of disturbed sensibility in the right hand or arm.

May 9, 1919, there was an extensive area (Fig. 5) of diminished touch and pain sensibility on the posterior surface of the left hand and arm, most marked on the forearm. May 28, 1919, the area was considerably diminished and could be outlined in anything like its original form only by stroking touch. There was no loss of appreciation of weight but two point discrimination was definitely lessened on the dorsum of the forearm and a small area of somewhat lessened appreciation of cold at 4 C. was found on the dorsum of the forearm a little below the elbow.

Case 4.—The patient while advancing was struck by shrapnel in the left upper arm and left leg, Nov. 1, 1918. His arm felt dead but he could move it at the elbow directly after receiving the wound. He could not move the fingers; the arm did not jerk or twitch. The next day he noticed a definite numb area on the back of his left hand, forearm and arm. He could move the arm at the shoulder but not at the elbow or wrist. The fingers could be feebly flexed.

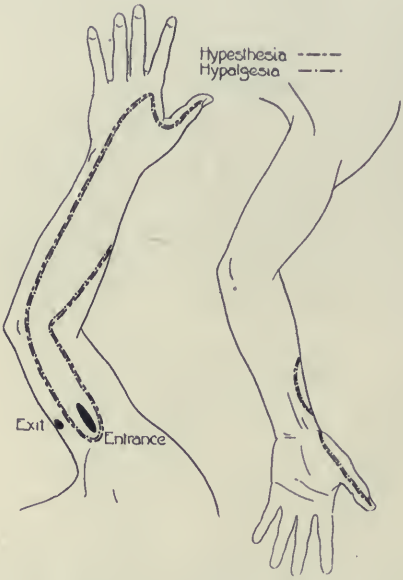

Fig. 5 (Case 3).—Condition, May 9, 1919.

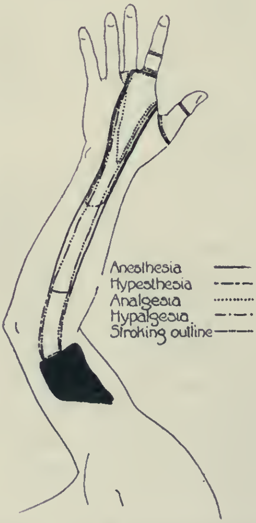

Fig. 6 (Case 4).—Area of sensory disturbance.

When examined May 13, 1919, he had a large scar, 6 by 2½ inches, over the lower end of the outer side of the left upper arm, adherent to the bone. The area of sensory disturbance (Fig. 6) extended down the back of the forearm in a rather narrow line and in the hand there was loss or great diminution of touch and pain over the first phalanx of the thumb but the skin over the first metacarpal and over the first interosseous space responded normally. In this case the loss of sensibility was more complete in the areas affected in the hand than in the forearm and in the lower part of the forearm than in the upper.

A further examination, May 28, 1919, showed an area of complete loss of sensibility to temperature of 5 C. and to ice over the region of greatest sensory loss on the hand, but on the thumb similar temperatures were fully appreciated. Joint sensibility was normal. Pressure sense was greatly diminished on the hand (200 gm.) and much less diminished on the forearm (27 gm.). On the hand, in two point discrimination, he gave 47 per cent. of correct answers at 40 mm. as against 53 per cent. at 22½ mm. on the right hand. On the left forearm he gave 53 per cent. of correct answers at 80 mm. and on the right forearm 53 per cent. of correct answers at 47 mm.

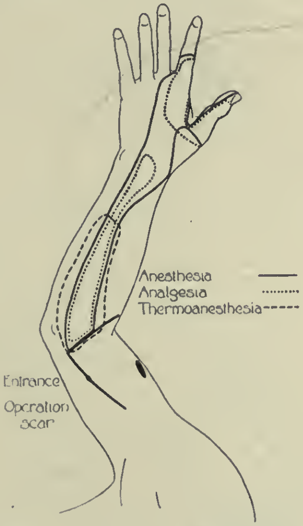

Fig. 7 (Case 5).—Area of sensory disturbance.

In July he went to the beach and both forearms became much sunburnt. The left was a little more affected than the right and almost blistered, but the two felt equally sore and hot.

Case 5.—Sept. 27, 1918, while walking, the patient was struck by a machine-gun bullet in the right leg and by a high explosive fragment in the left upper arm. The humerus was fractured. The left hand dropped immediately at the wrist and there was subsequently no movement at the wrist joint, though he could flex the fingers slightly. The hand and arm from the elbow down were numb on the dorsal surface, but he retained the sense of feeling on the palmar side and there was no pain except at the site of injury.

When examined, May 28, 1919, the ordinary line of numbness (Fig. 7) was found on the posterior surface of the forearm and thumb and the first and part of the second phalanges of the first finger were involved, but the skin

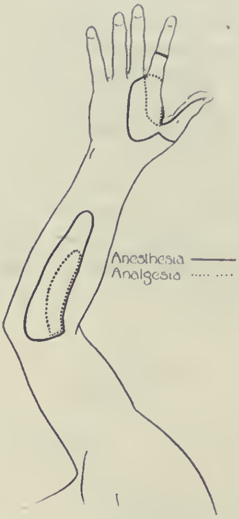

Fig. 8 (Case 5).—Condition, June 30, 1919.

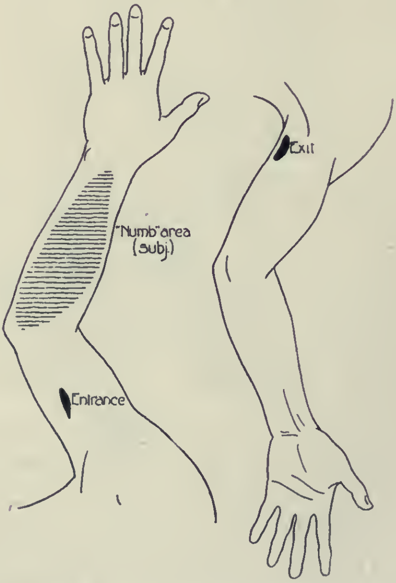

Fig. 9 (Case 6).—Area of subjective numbness.

over the first metacarpal and a part of the first interosseous space was unaffected. Pressure sense was much more affected in the hand and wrist than in the middle and upper part of the forearm, whereas two point discrimination was much more deficient in the upper part of the left forearm than in the wrist or hand. The area of loss to cold was wider than that of loss of touch and pain. There was no diminution of vibration or joint sensibility.

On June 30, 1919, the loss of sensation was about the same on the dorsum of the hand and upper two thirds of the forearm, but had quite disappeared over the wrist and lower one third of the forearm (Fig. 8).

Case 6.—Oct. 14, 1918, the patient, while standing, was struck by a fragment from a shell which exploded about 3 feet from him. He sustained a severe compound, comminuted fracture of the left humerus. He bled considerably and was unconscious for three or four days. His arm was heavily bandaged when he regained consciousness. His idea of the sensory loss was not exact, but he was sure that at least he had had numbness of the back of the left thumb, of the proximal part of the left index finger and did have feeling somewhere toward the ulnar side of the hand. There was complete wrist drop.

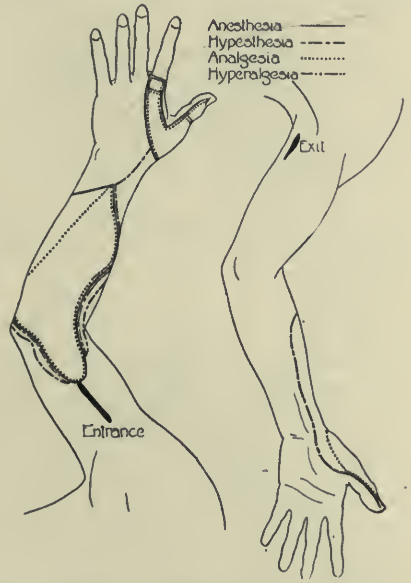

Fig. 10 (Case 6).—Area of sensory disturbance.

When examined, May 5, 1919, the area of subjective numbness (Fig. 9) included a large portion of the dorsum of the left forearm, but he was not aware of any loss over the hand. The examination showed (Fig. 10) a definite area of anesthesia and analgesia of the dorsum of the thumb and of the wrist and anesthesia of practically the entire dorsal region of the forearm and of the lower part of the upper arm with a less wide area of analgesia. On the volar surface of the forearm, toward the radial side, there was hypesthesia and over the outer side of the thenar eminence and thumb there was anesthesia and analgesia. There were small areas of hyperalgesia over the distal phalanx of the thumb and over the knuckle of the index finger.

The involvement of the radial side of the forearm, in this case, suggests the possibility that the musculocutaneous was injured, but it may at least be said

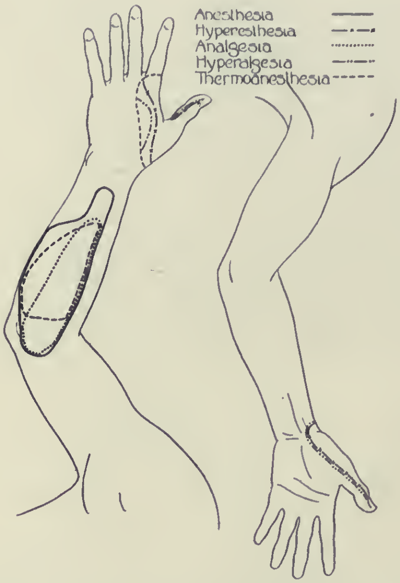

Fig. 11 (Case 6).—Condition, June 1, 1919.

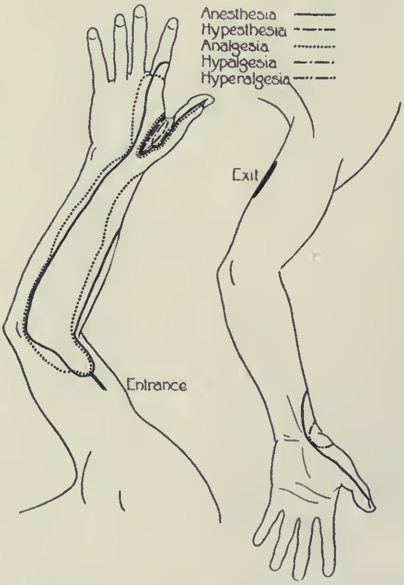

Fig. 12 (Case 7).—Area of sensory disturbance.

that the injury was not in such a position as to be likely to involve the musculocutaneous, unless it did so above its supply to the biceps and there was no evidence of any motor involvement to the biceps.

Examination, June 1, 1919, showed (Fig. 11) hyperesthesia and hyperalgesia and loss of cold at 5 C. over the area of the hand previously showing sensory loss, no evidence of any sensory involvement of the dorsum of the wrist and a decided diminution of the area on the forearm.

Case 7.—The patient was struck by a machine-gun bullet, Sept. 29, 1918, causing compound, comminuted fracture of the left humerus. Following the injury there was extreme pain in the whole arm and considerable hemorrhage. He does not recall whether there was loss of feeling or motion following the injury. He was operated on thirty-six hours later, and is sure there was no paralysis of motion following the operation. The arm was placed in a Thomas splint with extension. Three weeks later he noticed that wrist drop was developing and that the end of the thumb was growing numb. The loss of motion and sensation increased for a time, but he thinks that during the past few months there has been progressive improvement.

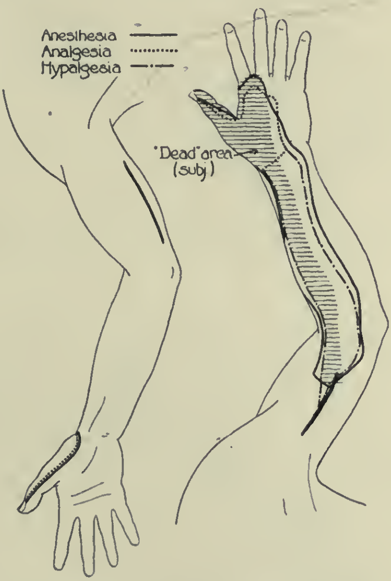

Fig. 13 (Case 8).—Area of sensory disturbance.

When examined, Aug. 19, 1919, a wide area of sensory change was found (Fig. 12). Sensibility was much less disturbed in a narrow line over the first interosseous space than in the areas to either side.

Case 8.—Sept. 30, 1918, while standing, the patient was struck on the outer side of the right upper arm and the bullet came out on the inner side. Directly following the injury, he had no power of sensation or motion in the right arm or hand. Seven hours after the injury he was operated on and the musculospiral nerve sutured. Just before the operation, he found he could flex the right fingers feebly. After the operation there was numbness down the outer side of the right arm, on the dorsum of the forearm and on the radial side of the hand.

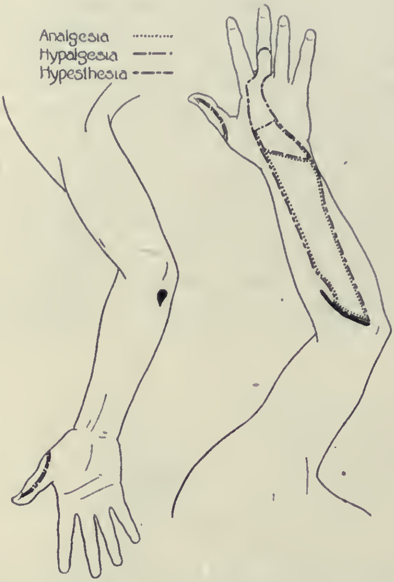

Fig. 14 (Case 9).—Area of sensory disturbance.

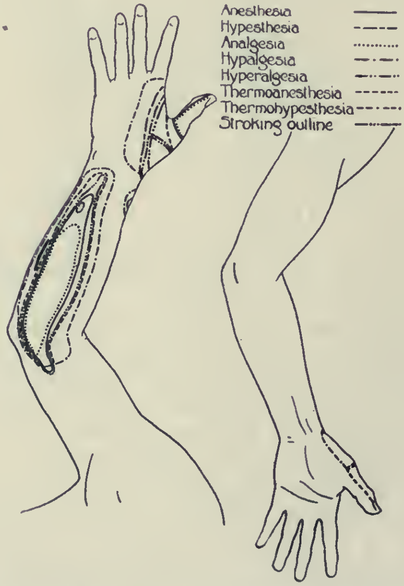

Fig. 15 (Case 10).—Area of sensory disturbance.

April 8, 1919, examination showed (Fig. 13) an extensive loss of sensation on the dorsum of the hand and forearm and on the outer side of the upper arm. Diminution of pain and touch was much more marked distally than proximally in the forearm and more marked in the hand than in the forearm or arm. Vibration and joint sensibility were normal but two point discrimination was diminished. The mental condition of the patient did not permit a very satisfactory examination.

Case 9.—Sept. 29, 1918, the patient was struck by a piece of shrapnel on the dorsal surface of the right arm just below the elbow. The fragment passed out through the ulnar surface. Directly after the injury was received the entire extremity from the elbow down felt numb and "tingled as if a nerve had been struck." He was operated on the following day, but thinks no nerve was sutured. Subsequently he found about the same area of numbness on the dorsum of the arm and hand as at present, except that it was slightly larger. At an examination made May 26, 1919, it was found that the loss of sensibility (Fig. 14) was much more complete on the dorsum of the forearm than on the hand, and there was an area of normal sensibility over the first metacarpal and the first interosseous space. There was no perceptible loss of sensation on the index finger, but the dorsum of the first phalanx of the second finger showed hypesthesia. Vibration sense was practically normal.

Case 10.—While lying down, Oct. 24, 1918, the patient was struck on the outside of the left upper arm by a machine-gun bullet. He felt "as if a hot poker were being run through it and the arm felt numb from the wound down." When dressed he could feel the bandages about the palm of the hand and the ulnar part of the wrist but not anywhere on the forearm. Feeling began to return in the forearm about the middle of November. When examined, June 4, 1919, there was a large area on the dorsum of the forearm (Fig. 15) of very marked sensory disturbance. Anesthesia was wider than analgesia. The margin of stroking outline lay outside both and the line of thermoanesthesia and of thermohypesthesia (8 C.) was still wider. The dorsum of the thumb showed anesthesia and analgesia, but the dorsum of the first metacarpal and of the first interosseous space showed hypesthesia and, over part of the former, hypalgesia. Vibration sense was reduced on the thumb. In two point discrimination he gave 53 per cent. of correct answers at 25 mm. in the right forearm and 53 per cent. of correct answers at 70 mm. in the left forearm.

Case 11.—The patient was struck in the left upper arm Nov. 7, 1918, by a machine-gun bullet which caused a compound, comminuted fracture at the middle of the humerus. There was immediate and complete motor paralysis of the musculospiral nerve, but he is uncertain as to the sensory condition. Examination, April 24, 1919, revealed anesthesia and analgesia (Fig. 16) of the area on the second metacarpal and the second interspace and hypesthesia and hypalgesia on the dorsum of the thumb and the first metacarpal with a narrow band of normal sensibility between them. There was also hypesthesia and hypalgesia over a band of considerable width extending down the dorsum of the forearm. June 21, 1919, the area on the forearm was considerably reduced. The condition of the hand was not much altered except for hyperalgesia over the thumb and an increase in the size of the normal band over the first interosseous space. Aug. 16, 1919, no sensory loss could be discovered in the forearm, and the hand was greatly improved (Fig. 17).

Case 12.—While the patient was asleep, Oct. 15, 1919, a bullet passed through the left forearm, just below the elbow. Directly the entire extremity was numb from the elbow down. Feeling later returned in all parts except in a line down

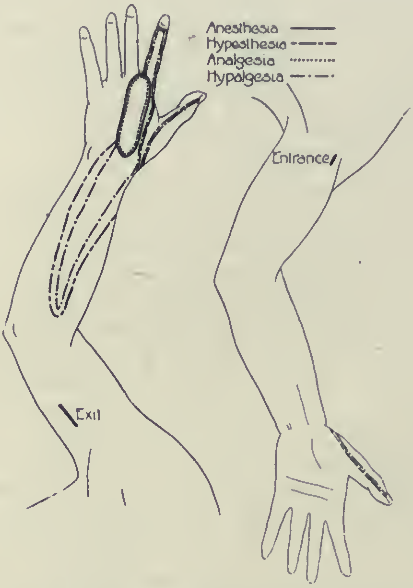

Fig. 16 (Case 11).—Area of sensory disturbance.

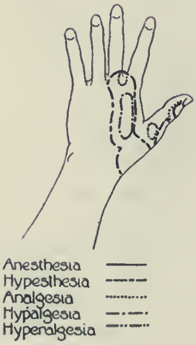

Figure 17

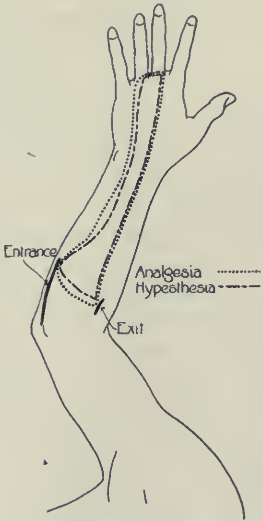

Figure 18

Fig. 17 (Case 11).—Area of sensory disturbance, Aug. 16, 1919.
Fig. 18 (Case 12).—Area of sensory disturbance.

the center of the back of the hand and forearm. Examination, July 10, 1919, showed hypesthesia and analgesia in the area shown in Figure 18 and a partial paralysis of the musculospiral motor supply.

Case 13.—Nov. 1, 1918, the patient was struck on the inside of the left upper arm by a bullet which was removed on the upper side of the upper arm. So far as the patient knows, there was no motor or sensory loss following this operation, but about March 1, 1919, a sequestrum was removed from the upper arm and subsequently he had difficulty in extending the fingers and thumb. When examined, June 25, 1919, there was still no extension of the thumb or fingers of the hand, but extension of the wrist was fair. On the dorsum of the forearm was an area 1¾ by 1½ inches (Fig. 19) of loss of sense of touch. In this area pin prick was described as "less sharp," but as "hurting more," with a feeling of being different from a normal pin prick. No other sensory disturbance could be found.

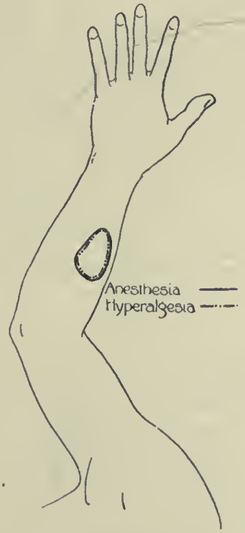

Fig. 19 (Case 13).—Area of sensory disturbance.

There were twenty-seven cases in which the sensory phenomena, due to injury of the musculospiral nerve, were limited to the hand or to a region extending slightly above the wrist. Of these, fourteen were selected for reporting.

Case 14.—While riding a motorcycle, Nov. 9, 1918, the patient's left arm dropped suddenly to his side. He was not conscious of having been injured, and there was no pain or jerking of the hand or arm. An examination showed he had been struck in the left arm by a bullet. He could flex his fingers, and he is sure that the dorsum of the thumb and of the first finger and the space between were numb and that he could not appreciate pin prick in this region as pain.

When examined, April 17, 1919, only an area on the dorsum of the first phalanx of the thumb and a small area on the outer surface of the dorsum of the first phalanx of the index finger felt numb. Examination showed (Fig. 20) analgesia to 12 gm. of pressure over the knuckle of the index finger and to

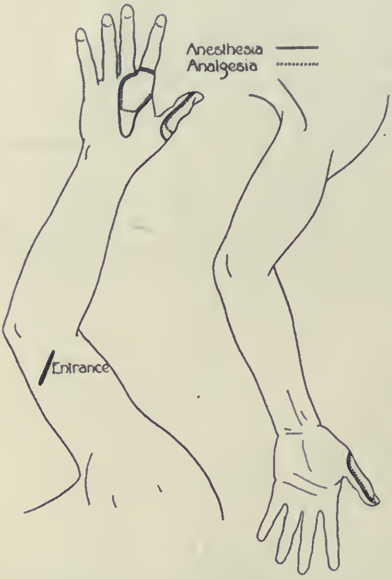

Fig. 20 (Case 14).—Area of sensory disturbance.

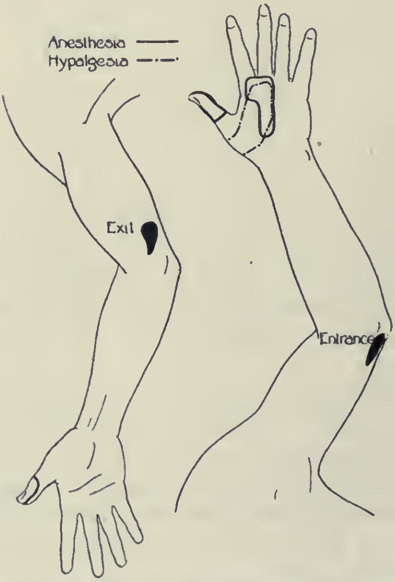

Fig. 21 (Case 15).—Area of sensory disturbance.

10 gm. of pressure over a band extending back about half way to the wrist. The dorsum of the first and second phalanges of the thumb was analgesic to 12 gm. of pressure. Both areas were anesthetic to touch as was also an area extending proximally from the knuckle almost to the wrist. He gave two point discrimination in 53 per cent. of tests over the normal hand at 21¼ mm. and in 53 per cent. of tests over the affected area at 47 mm. On the area over the knuckle, he could barely discriminate 25 gm. of pressure. With the exception of this region, discrimination of pressure was not greatly reduced in the affected area.

Case 15.—The patient was injured Oct. 3, 1918, by a machine-gun bullet entering the right arm at the elbow. He was sitting with his elbow on his knee. He felt as if a "log had struck him" and was knocked over but did not become unconscious. There was no twitching or jerking in the hand or arm. The dorsum of the thumb and of the back of the hand (with the exception of the extreme ulnar side) was numb. He cannot recall anything concerning the feeling in the back of the fingers, but does not remember any numbness, and he is positive there was no numbness in the forearm. There was some stinging feeling in the back of the hand for a time, but this had disappeared at the examination on April 18, 1919, when he showed an area of anesthesia and analgesia as outlined in the diagram (Fig. 21).

A second examination on June 10, 1919, showed some improvement in sensation. Vibration and joint sensibility at this time were normal. Cold at 5 C. was readily felt. In discrimination of two points he gave 53 per cent. of correct answers at 45 mm. in the right hand and 100 per cent. at 26 mm. in the left hand.

Case 16.—Aug. 10, 1918, while lying flat on his face, the patient was struck by a fragment of shell which entered the left arm 2 inches below the elbow on the radial side and passed out through the posterior surface of the arm just above the elbow, fracturing the humerus. Directly after he was struck, the hand and arm drew up and the "fingers clenched," after which the entire arm became limp and numb. He felt as though the arm from the elbow down did not belong to him, and he could make no movement from the hand or forearm.

When examined, May 23, 1919, there was an area (Fig. 22) of anesthesia and hyperalgesia over the phalanges of the thumb and of anesthesia and analgesia over the second metacarpal bone and second interspace and over most of the first interspace. Over the first metacarpal bone there were hypesthesia and hypalgesia. Sensibility to cold at 2 C. was lost in all this area and over a considerable area over the thenar eminence. Cold at 22 C. was lost over a somewhat wider area. The threshold for two point discrimination was about one-half higher in the affected arm than on the unaffected side. Weight discrimination was very little changed. In the first, second and third fingers there was an area of uncertain loss to touch, probably due to local skin condition.

Case 17.—July 16, 1918, a fragment of shell entered over the lower end of the left biceps and passed out a little above the outer condyle of the humerus. When examined, May 26, 1919, there was anesthesia (Fig. 23) over the entire dorsum of the thumb and over the radial side of the dorsum of the hand with the exception of a band about 1 inch wide extending down the first interspace. Pin prick was appreciated as such at from 4 to 8 gm. of pressure; two point and weight discrimination were both only slightly impaired in the same region. There was no loss of joint or vibration sensibility. An area of hyperalgesia was found over the web of the thumb.

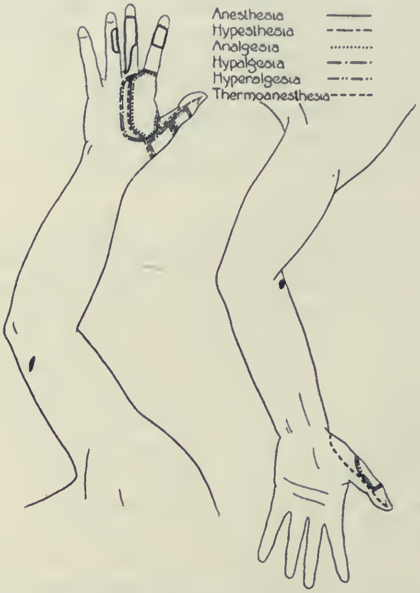

Fig. 22 (Case 16).—Area of sensory disturbance.

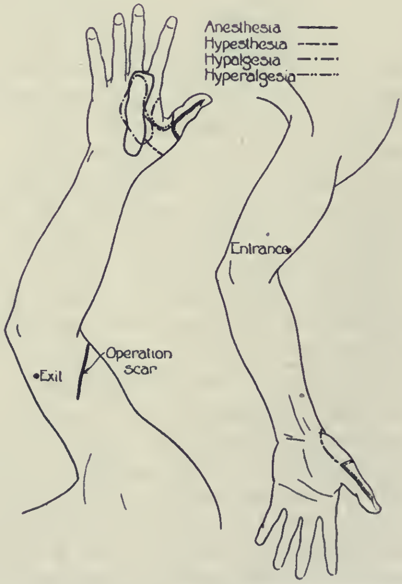

Fig. 23 (Case 17).—Area of sensory disturbance.

Case 18.—The patient was injured in the left arm, on the outer side, just above the elbow by a piece of shrapnel, Oct. 4, 1918. He was knocked down, and remained unconscious for a few minutes. On recovering, he felt as though the arm had been blown off. When he attempted to move it, it hung limply by his side, and he said it "felt as if it weighed a ton." The next morning there was numbness in the area shown in the diagram and over the dorsum of the first finger. He is positive there was no numbness in the arm. He noticed improvement first in the distal part of the forefinger and then in the region of the first metacarpal.

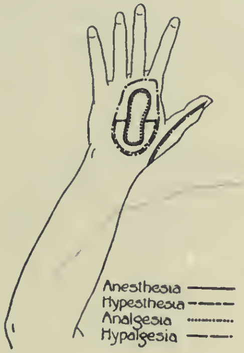

Fig. 24 (Case 18).—Area of sensory disturbance.

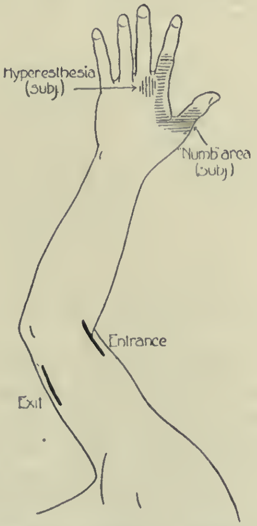

Fig. 25 (Case 19).—Area of sensory disturbance.

When examined, May 12, 1919, a roentgenogram showed some small fragments of bone still in the wound and in the region of the musculospiral nerve. There was an area of anesthesia to touch and analgesia to prick over the second and third metacarpals as shown in Figure 24 and outside of this there was an area of hypesthesia and hypalgesia. The dorsum of the phalanges of the thumb and of the distal part of the first metacarpal was anesthetic and analgesic. In the first interosseous space was an area of normal sensibility.

Case 19.—Oct. 29, 1918, the patient was struck in the left arm just to the outer side of the elbow. The missile passed out at a slightly higher level on the posterior surface of the upper arm, fracturing the humerus. His arm was immediately paralyzed from the elbow down and subsequently there was an area of "numbness" (Fig. 25) involving the dorsum of the first phalanx of the thumb, the distal part of the first interosseous space, a band over the second metacarpal and the first phalanx of the forefinger. Over and just proximal to the knuckle of the second finger was a circular area of subjective hyperesthesia. Examination, May 16, 1919, showed (Fig. 26) hypesthesia and hypalgesia over the first phalanx of the index finger, analgesia and hypesthesia over the thumb and anesthesia and analgesia over the remainder of the area. The subjective hyperesthetic area gave normal response to touch and prick.

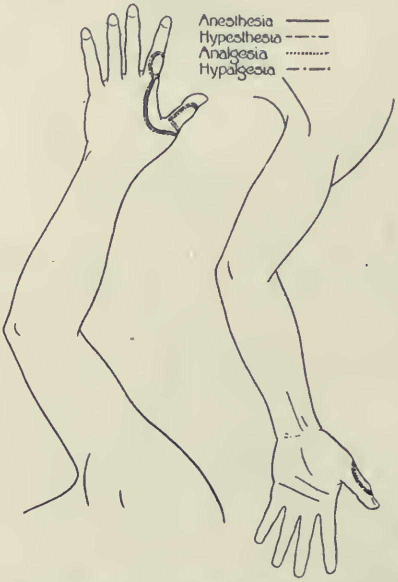

Fig. 26 (Case 19).—Condition, May 16, 1919.

Case 20.—The patient was injured Oct. 4, 1918, by a machine-gun bullet. He was lying down, and the bullet entered at the lower part of the right deltoid on the outer surface, and came out on the inside of the arm just below the axilla. The whole arm felt numb subsequently, and motor power was completely lost. After a few hours the arm was less numb and began to feel "sore" but not in any special locality. At the same time the numbness largely disappeared from the upper extremity but for three weeks there was some loss of feeling in the forearm, not clearly localized. There has been no numbness since. After this there was not much change in feeling but marked improvement in motion.

When examined, May 13, 1919, he outlined an area of numbness on the back of the hand a little smaller than that found on examination and an area on the inside of the hand considerably greater than could actually be demonstrated. Examination showed (Fig. 27) anesthesia and analgesia over the dorsum of the phalanges of the thumb, over the first and part of the second

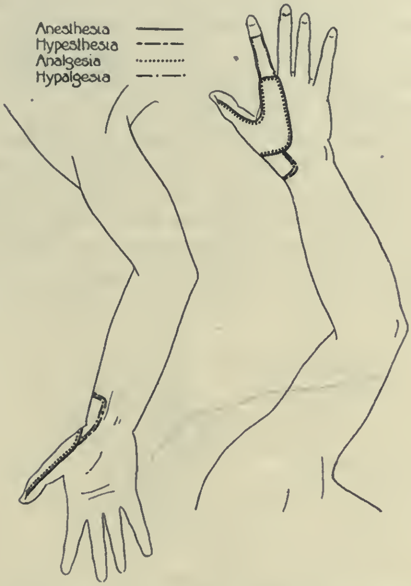

Fig. 27 (Case 20).—Area of sensory disturbance.

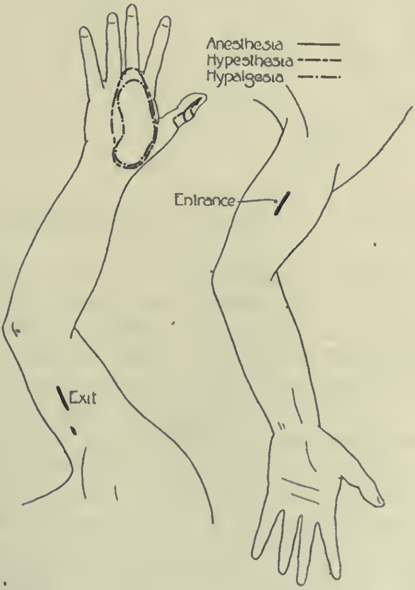

Fig. 28 (Case 21).—Area of sensory disturbance.

metacarpals, including the first interosseous space, with an area of hypesthesia and hypalgesia considerably wider. There was also hypalgesia over the first and second phalanges of the right index finger.

Case 21.—The patient was hit by a machine-gun bullet, Oct. 16, 1918, in the upper part of the left arm, and the humerus was fractured. He felt no pain and did not know that he was hurt, until his arm, on which he was leaning, gave way. For one week the arm felt numb up to the shoulder and after three weeks there was no sensory loss of which the patient was aware other than the area shown on examination, June 5, 1919 (Fig. 28). The area of hypesthesia on the dorsum of the hand was wider than the area of hypalgesia. The dorsum of the second phalanx of the thumb was anesthetic and hypalgesic. The dorsum of the distal half of the first phalanx was anesthetic and of the proximal half hypesthetic, but he could feel pin prick in both places.

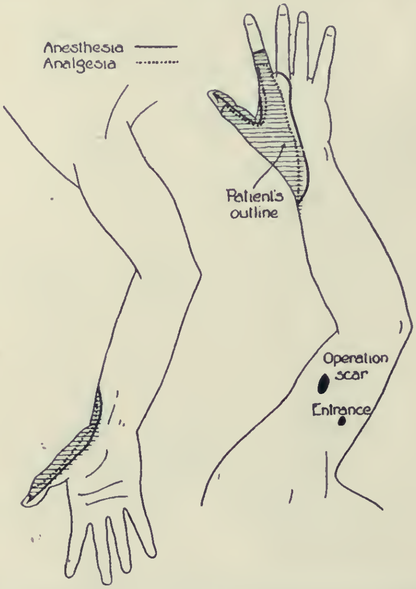

Fig. 29 (Case 22).—Area of sensory disturbance.

Case 22.—The patient was injured, Nov. 1, 1918, in the right arm and forearm. The only part of the early history available, was that after the injury was received he had a wrist drop. When examined, May 2, 1919, he had slight extension of the wrist, a very slight extension of the fingers and none of the thumb. There was anesthesia and analgesia (Fig. 29) of the dorsum of the thumb, of the first phalanx of the index finger and of the radial side of the hand, extending upward a little distance above the wrist. Anesthesia was somewhat wider than analgesia. The area of stroking change was slightly larger than either and the subjective area was somewhat smaller. Sensation was preserved in a region along the edge of the web of the thumb.

June 9, 1919 (Fig. 30), sensation had improved decidedly. The dorsum of the thumb and of the second metacarpal remained anesthetic and analgesic but the area between was very slightly hypalgesic. Loss of sensation of cold at 8 C. and the stroking outline almost coincided. On the front of the right wrist was a region of increased sensibility to cold at 8 C.

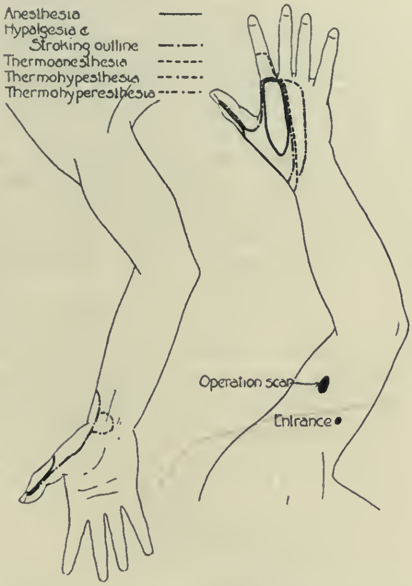

Fig. 30 (Case 22).—Improvement in sensation, June 9, 1919.

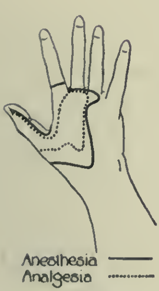

Figure 31

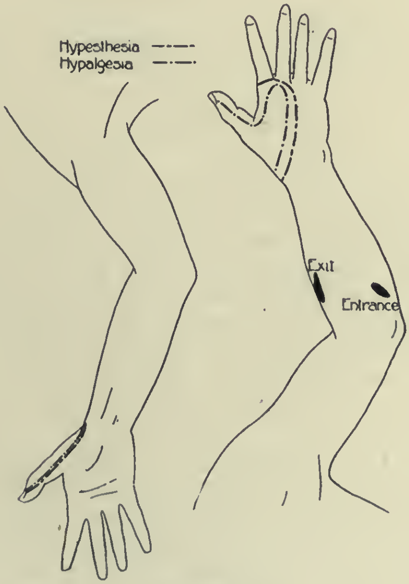

Figure 32

Fig. 31 (Case 23).—Area of sensory disturbance.
Fig. 32 (Case 24).—Area of sensory disturbance.

Case 23.—The patient was struck by shrapnel, Oct. 9, 1918, in the lower one-third of the right upper arm, receiving a compound, comminuted fracture of the humerus with considerable loss of bone. When examined, May 16, 1919, there was considerable loss of substance of the shaft of the humerus, a complete motor paralysis of the musculospiral supply and an area of anesthesia and analgesia on the dorsum of the hand (Fig. 31).

Case 24.—Oct. 14, 1918, a bullet passed through the right arm, just below the elbow, resulting in musculospiral paralysis. The history of the early symptoms is lacking. When examined, May 22, 1919, there was (Fig. 32) hypesthesia and hypalgesia of the dorsum of the thumb and of the radial side of the hand. Extension at the wrist was poor and of the thumb and fingers was entirely lacking.

Case 25.—The patient was shot through the left elbow, Oct. 12, 1918. Musculospiral paralysis resulted. The early history was not available. When examined, June 30, 1919, there was fair extension of the wrist, fingers and thumb, but there was a large area of anesthesia and analgesia on the dorsum of the hand and an area of hypesthesia and hypalgesia on the dorsum of the thumb (Fig. 33).

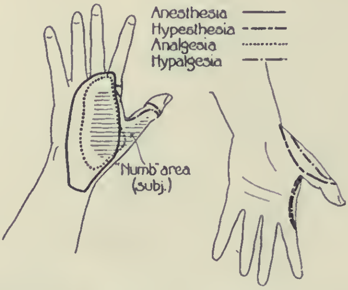

Fig. 33 (Case 25).—Area of sensory disturbance.

Case 26.—The patient was struck on the outside of the right arm just above the elbow, Oct. 8, 1918, by a machine-gun bullet which came out on the posterior and inner surface at about the same level. Motion was lost at once in the musculospiral supply and for a time the arm felt numb, but he thinks he could distinguish touch at all times. He recognized no complete loss of sensation. When examined, June 5, 1919, there was a small area of anesthesia (Fig. 34) about the knuckle of the right index finger and on the dorsum of the second phalanx of the thumb with hyperalgesia in both areas and inability to extend the thumb. There was also rather weak extension of the fingers.

Case 27.—The patient was injured Oct. 8, 1918, by high explosive fragments which entered the left upper arm and forearm. The arm at once became limp and he felt as though he had lost it. There was no sensation at any point on the arm until the following day, after which sensation returned except in the area shown in the diagram. When examined, April 24, 1919 (Fig. 35), the entire dorsum of the thumb was anesthetic and there was a small area of analgesia on the distal part of the dorsum of the first phalanx.

Case 28.—Oct. 15, 1918, the patient was struck by high explosive fragments at various points, including the front of the right axillary fold. The entire right arm and hand were paralyzed for motion and sensation for some hours

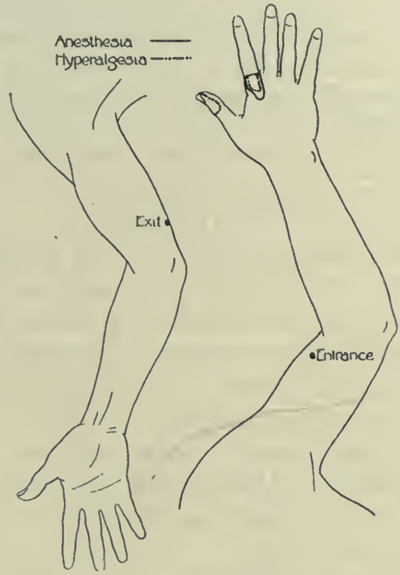

Fig. 34 (Case 26).—Area of sensory disturbance.

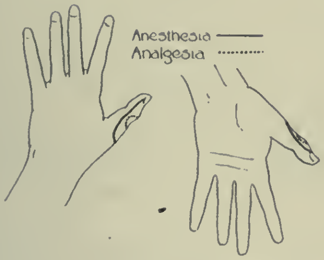

Figure 35

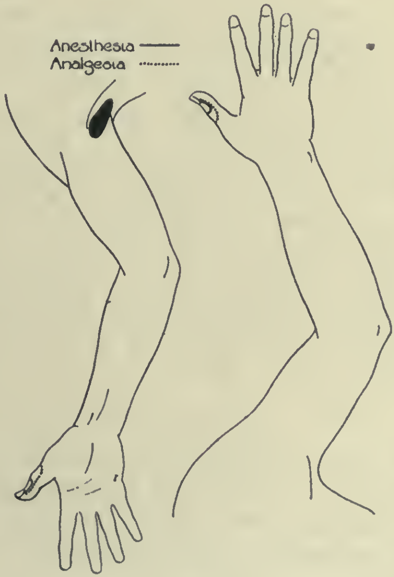

Figure 36

Fig. 35 (Case 27).—Area of sensory disturbance.
Fig. 36 (Case 28).—Area of sensory disturbance.

with subsequent gradual improvement. After one week the only sensory loss was over an area on the thumb about as large as at present. A musculospiral motor paralysis persisted for three months but at present his only marked motor loss is his inability to extend the thumb and index finger.

When examined, June 30, 1919, there were analgesia of the dorsum of the first and second phalanges of the thumb and anesthesia of the dorsum of the second phalanx (Fig. 36). No other sensory loss could be elicited.

SENSORY PHENOMENA

The literature in existence in reference to the sensory phenomena in musculospiral injuries is surprisingly limited, but in a general way there appears to be an agreement that the musculospiral is chiefly a motor nerve and that the sensory changes resulting from its injury are limited in extent and unstable in their location. Head and Sherren[2] even state, "It has long been known that division of the musculospiral in the region of the spiral groove causes no definite loss of sensation over the thumb or back of the hand," and work on the hand and arm was done by Head after division of the external cutaneous nerve as well as of the superficial radial.

Borchardt[3] reports seventeen cases of injury of the musculospiral nerve. In six instances there is no statement as to sensory deficiency. In three cases there was definite sensory change and in the other eight cases the sensory condition is dismissed with such statements as: "Nothing of consequence in the way of sensory loss," "no true sensory disorders," or "a dull sensation in the fingers."

Tinel,[4] in speaking of the subject, says: "Anesthesia in musculospiral paralysis is not often found anywhere else than on the hand. Seldom does it reach the typical distribution of the musculo-spiral, far more seldom does it go beyond it. Most frequently it limits itself to a very restricted part comprising the dorsal region of the first and the second metacarpal. It is often very slight, sometimes even it can scarcely be seen; in any case, it is exceptional to find complete anesthesia in this region; at most we have more or less marked hypesthesia."

Byrne's case[5] showed both epicritic and protopathic loss over the dorsum of the thumb, part of the thenar eminence, the radial half of the dorsum of the hand and the lower wrist.

My own observations lead me to believe that though a lesion of the musculospiral nerve at any point produces less sensory disturbance than one would expect considering its anatomic distribution, it is rarely, if ever, that some definite area of sensory loss may not be found if carefully sought.

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[2] Head and Sherren: Brain **28**:177, 1905.

[3] Borchardt: Bruns Beiträge **97**:253.

[4] Tinel: Nerve Wounds. 1918, p. 107.

[5] Byrne: J. Nerv. & Ment. Dis. **43**:489, 1916.

Sensory phenomena, as appreciated by the patient, are not pronounced and are limited in extent. Usually he will tell of an area of numbness over the dorsum of the thumb and especially of the first phalanx, over the first interosseous space and perhaps over the first phalanx of the index finger and more or less over the distal portion of the second metacarpal. The area on the back of the forearm is often ignored even when by examination a large area of well defined sensory change can be demonstrated. In the one instance of well marked sensory change on the back of the upper arm that came under my observation, this was recognized by the patient. A possible satisfactory explanation for the failure of the patient so many times to recognize his sensory loss may be that the musculospiral nerve, limited in sensory fibers, supplies only epicritic and protopathic sensibility to the integument and that deep sensibility is supplied by other nerves. The patient, depending largely on some form of irritation that gives a response through deep sensibility, for his consciousness of a stimulus, fails to recognize the sensory loss until it is shown him by adequate methods of examination.

In any case, when sensibility is to be tested in musculospiral injury, the skin should be shaved, as was done in all the cases reported, and tactile sensation tested with cotton, wool or a camel's hair brush with the lightest possible touch. Pin prick is usually lost as pain in a similar or somewhat smaller area, but is ordinarily recognized as pressure since, as already stated, deep sensation is not commonly lost in musculospiral injury. Sensation to cold was usually impaired or lost in an area corresponding more or less to the loss for touch and prick and sometimes in a larger area. In some cases the failure to show in the diagram any loss of sense of cold is so out of keeping with the other phenomena that it can be explained only by lack of care on the part of the examiner or by lack of intelligence and attention on the part of the patient. In only one instance was there any excessive response to cold.

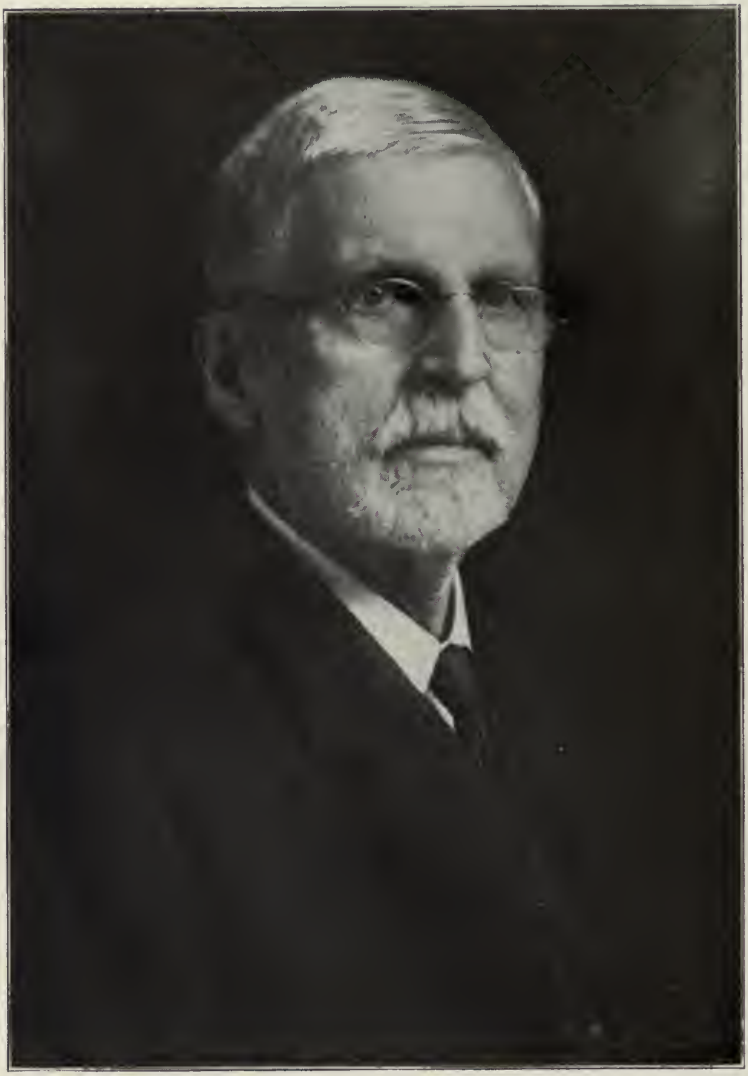
Sense of the position of the joints was not lost or notably impaired in any of my cases, but vibration sense was often impaired though not lost, especially on the thumb. Discrimination of two points as two was much diminished in most of the cases, but appreciation of weight was not greatly reduced even in cases with well marked loss of superficial sensibility.

One of the problems which I encountered was to determine the sensory loss in the forearm which can fairly be ascribed to the musculospiral nerve. In several of my cases in which there was no evidence that the musculocutaneous nerve had been severed there was a marked loss of sensation over the area usually ascribed to that nerve. In two instances nearly all of the dorsum of the forearm was numb, but the volar surface was not involved. In such a case it is as reasonable to assume that because the volar surface was little or not at all involved the sensory loss could not be ascribed to any injury to the musculocutaneous or the internal cutaneous nerve as to assume that because the dorsal portion of the area usually ascribed to these nerves was involved, the nerves themselves must be assumed to be involved in the injury. In such cases as Cases 7, 8 and especially 12, it may fairly be doubted that the area of sensibility loss represents only musculospiral involvement; however, it must be understood that no case was included in this series in which the motor phenomena did not clearly demonstrate musculospiral lesion or in which the motor phenomena showed any additional material lesions. A full description of these motor phenomena could only have lengthened this paper.

In those cases in which some degree of numbness was found in the forearm, there was often an area over the dorsum of the wrist in which sensation was more nearly normal or wholly normal. In ten instances in the series reported there was a strip passing from the web between the thumb and first finger in a proximal direction, of normal reaction or of very slight sensory loss separating the more deeply affected area over the dorsum of the first and second metacarpals, respectively.

As a rule, loss of sensation, if present in the thumb, extended down to the nail, but in a few instances there was a narrow band of retained sensibility proximal to the nail, and it was a common occurrence to find the dorsum of the first metacarpal less involved than the phalanges of the thumb. On the first phalanx of the thumb loss of sensibility was ordinarily bounded by the hair line on the median side, but on the external side it extended over somewhat on the palmar surface. This was much more marked over the thenar eminence where the loss of sensibility extended in a curved line on to the palmar surface. Where no evidence of this loss over the thenar eminence appears in a number of the diagrams the explanation probably lies in a lack of care on the writer's part in recording the result of the examination rather than in its actual failure to appear. Where loss of sensibility existed in the area between the thumb and first finger and between the first and second fingers the line of demarcation extended well beyond the hair line and almost to the edge of the web.

I wish to acknowledge my indebtedness to Miss Ethel Cornell, Ph.D., and to Miss Margaret Stubbs for their very valuable assistance in the examinations which form the basis of this paper.



JAMES JACKSON PUTNAM
1846-1918

JAMES JACKSON PUTNAM

HIS CONTRIBUTIONS TO AMERICAN NEUROLOGY

E. W. TAYLOR, M.D.
BOSTON

Dr. Putnam was born in Boston, October 3, 1846, the son of James Gideon and Elizabeth Cabot (Jackson) Putnam, a distinguished heritage on both sides with the very best New England traditions of loyalty and good citizenship. His paternal grandfather, Samuel Putnam, was for many years a judge of the Supreme Court of Massachusetts; his father was a physician of distinction, and his mother a daughter of James Jackson, one of the most notable figures of his period in American medicine, an appreciative biography of whom Dr. Putnam published in 1905.

Dr. Putnam graduated from Harvard College in 1866, at the age of 20; he stood high in his classes, and already gave promise of the attainments of his later years. After leaving the Harvard Medical School, and completing his apprenticeship at the Massachusetts General Hospital, he continued his medical studies in Leipzig and Vienna, under the tutelage of Rokitansky and Meynert. Paris and England were included in his tour of study, and at this time he came into intimate contact with Huylings Jackson, who doubtless exerted a strong influence on his development, and for whom he always entertained the warmest admiration. On his return to Boston he entered on his chosen work with the zeal and enthusiasm of a pioneer, and at once identified himself with the almost untried field of nervous disease. His first appointment at the Harvard Medical School was as lecturer on the application of electricity in nervous diseases—a strange title, illustrative of the conservatism of the time; two years later his title was changed to lecturer on diseases of the nervous system. In 1875, he was appointed clinical instructor; in 1885, instructor, and in 1893, professor of diseases of the nervous system—a position which he retained until his retirement by reason of age in 1912, when he was made professor emeritus. During this long period of activity, he taught and worked unceasingly, chiefly at the Massachusetts General Hospital, establishing there one of the first neurological clinics in the country, ably assisted for many years by Dr. George L. Walton. There were frequent discouragements; the importance of the subject was slow of recognition; the facilities for work were inadequate; prejudices, not yet wholly overcome, were many and disturbing. Dr. Putnam, as one of his contemporaries has recently expressed it, was not a "fighter," but in spite of discouragements the clinic grew; patience and an unfailing belief in the importance of his work brought results which mere aggressiveness might well have failed to accomplish. By degrees men were attracted to the clinic, and at the time of his death he had the satisfaction of knowing that his early effort had developed into a dignified department of essential and recognized value in the hospital organization.

During the earlier years, after his return from Europe, finding a lukewarm interest in the pathologic anatomy of the nervous system, he established a neuropathologic laboratory in his own house, and there, with an enthusiasm which atoned for lack of proper facilities, he accomplished much of his early pathologic work. This humble laboratory may well be regarded as the forerunner of the present endowed neuropathologic department at the Harvard Medical School. At this time, Dr. Putnam was also much interested in the physiology of the nervous system, in which he found a ready sympathizer in his friend, the late Dr. Henry P. Bowditch, for many years professor of physiology at the Harvard Medical School. His interests were wide, his capacity for work insatiable, and for nearly fifty years his pen was as active as his mind. His papers on a wide diversity of topics within his chosen field were never superficial, and never written merely for the sake of publication. He felt that he had a message, and that it was one of his functions to promulgate knowledge of the nervous system and its diseases in a somewhat unreceptive profession. He wrote assiduously and even laboriously; he was rarely satisfied with the ultimate product, but a reading, especially of his later papers and his addresses, can leave no doubt that he was a rare master of good English. His interest in the classics and in the best literature, together with his general scholarly instincts, combined to give his publications a literary excellence, too often lacking in technical writing. He was peculiarly happy and sympathetic in his biographical sketches, which his natural loyalty to his friends and his keen appreciation of fine qualities of character frequently prompted him to write. Weir Mitchell, Wadsworth, Bowditch and Folsom are some of those whose life-work he gracefully commemorated.

As a teacher he was less successful than as an investigator and clinician. This arose from a certain lack of clarity of expression and an overconscientious attempt to impart to elementary students the profundity of his own knowledge. The result was a failure at times to differentiate essentials from the more unimportant details, which led to a certain natural confusion in the student's mind. To those of more experience in the subject, he was always helpful, stimulating and suggestive.

It is fitting to review in some measure his contributions to medicine and neurology, in spite of the fact that too little time has elapsed to appraise with any degree of justice the full significance of his later work. A characteristic which stands out prominently was his progressiveness. He never stagnated; interest in the past lay for him in its significance for the future; his zeal for progress and his optimistic hope in new discovery were the keynotes of his energetic life and character, and these qualities are distinctly reflected in his published work. He was almost childlike, and at times possibly unjudicial, in his eagerness to incorporate new ideas into the timeworn problems of disease. This openmindedness, more often seen in youth than in maturity, seemed to increase with the passage of the years and constituted one of the unique charms of his personality.

Fresh from his contact with the leading minds of Europe, his earlier work was concerned largely with matters which were then in the forefront of investigation: localization, the physiology of the cortex, mechanical therapy, animal experimentation, disorders of the peripheral nerves, syphilis in relation to tabes and the like. To all of these subjects he made contributions of value. In 1879, he took up the cudgels for women, who were then under a storm of criticism at Zurich, where they had been admitted to the study of medicine. Much as he disliked controversy, he was a vigorous champion of what he regarded as principles, and then as always he stood on the side of liberality, and took sharp issue with those who saw disaster in the higher education of women and the moral catastrophe which coeducation was assumed to entail. The same spirit was later shown in the vivisection agitation, into which he entered with natural reluctance, but with fixed determination to uphold its justice and necessity.

The first significant scientific contribution was his paper published in 1881, on "Paresthesia of the Hands," probably the earliest adequate description of a condition which has since become generally recognized and incorporated in the textbooks. A few years later he published one of the first descriptions of adult poliomyelitis with the pathologic findings. At this period also he became interested in the problem of lead poisoning with its accompanying neuritis, and prosecuted a long series of researches, which did much toward establishing this important subject on a firm scientific basis. A little later he took up the arsenic question, and pointed out the hidden danger of poisoning from this source. Wall papers were incriminated, whether rightly or wrongly may perhaps be doubted, but this research excited wide interest and something approaching a panic, at least in the vicinity of Boston. In general, it will be acknowledged that this vigorous campaign in industrial disease was a contribution of distinct value and a precursor of the widespread agitation at present in the focus of attention.

Passing over many papers of minor importance, another definite contribution was the paper published in 1891 on "System Diseases of the Cord," a subject to which Dr. C. L. Dana had almost at the same time devoted attention, and which very properly is associated with the names of both investigators. This again was an original contribution of far-reaching importance with which American neurology should be credited. Myxedema, acromegaly, exophthalmic goiter and the problem of the internal secretions in general were the subjects of various papers, which are of great interest as foreshadowing present day researches. They are all illustrative of Dr. Putnam's tendency to be the first in the field and to play his part in the elucidation of new problems as they arose. In 1895, we find a paper entitled "Remarks on the Psychical Treatment of Neurasthenia," which seems to mark the beginning of his absorbing interest in the psychotherapeutic movement. In this paper, he made an ardent appeal for the recognition of the psychical element in the treatment of what we have since termed the psychoneuroses, but it was many years before he devoted himself almost exclusively to this field of research. Neuralgia of the trigeminal nerve, migraine, hyperostosis cranii, tumors, encephalitis, splanchnoptosis, hydrotherapy, epilepsy, the traumatic neuroses and the surgery of jacksonian attacks are some of the many subjects which from time to time occupied his attention. These papers were all carefully prepared, and helped in greater or less degree in clarifying the unsettled problems of etiology and treatment. In passing, two notable addresses should be mentioned: one, the Shattuck Lecture before the Massachusetts Medical Society in 1899, with the suggestive and happy title, "Not the Disease only, but also the Man," a charmingly written paper embodying the full duty of the physician, as a healer of the mind as well as of the body; the other, a thoughtful contribution to the broader aspects of neurology, read at the International Congress at St. Louis in 1904, entitled, "The Value of the Physiological Principle in the Study of Neurology."

Although the subject of structural neurology occupied a large part of Dr. Putnam's active professional life, he always had the warmest interest in the more subtle problems of the so-called functional disorders. His mind was naturally analytical and philosophical, and he had an overmastering desire to formulate what might be termed a practical metaphysics. "Physics," he said, "can come to its rights only through metaphysics." The attempt to work out a theory of life which should make a rational appeal to the scientific imagination and yet satisfy the fundamental demand for a moral conception of the world was the keynote of his later work. He was doubtless much influenced in his philosophical views by his friends William James and Josiah P. Royce, and by the writings of the German schools of philosophy, from Kant on, and also by Bergson, whom he was always fond of quoting. It is, then, not unnatural that he should have been strongly attracted by the work of Freud and his associates, which made an immediate and insistent appeal; for the last ten years of his life, as his papers, about twenty-five in number, attest, he gave himself over with his characteristic enthusiasm to the task of reconciling the materialism, or pessimism of Freud, as Southard would have it, with his own unquenchable optimism and with the moral purpose of the world, in which he so thoroughly believed. When the heat of controversial discussion has cooled and his papers are read without prejudice and in the spirit in which they were written, it is my belief that a contribution of no small value to the questions which have always vexed the minds of thinking men will be disclosed. In estimating these last years of his life, it is well for us to realize that he was concerned with no small issues, that he was striving with such help as he could get to work out a satisfactory theory of life in which the conflicting claims of exact knowledge and of philosophy and religion might in some degree be brought into accord—a difficult and perhaps impossible task, but one altogether worthy of the best endeavor of thoughtful men. His last published article, on which he was working at the time of his death, he had entitled, "Strength and Weakness in Psychoanalytic Doctrines." Concerned as he was with what he regarded as the unjustifiability of applying the doctrine of causality in the realm of human thought, as in the realm of physics—a basal principle of the Freudian conception — he says:

> But whatever one may say on this point, it is certain that the chance, made possible by researches of Freud and his colleagues, to substitute knowledge of some sort for unreflective emotional reaction, and thus to eliminate passion, misunderstanding and misery, even if only in somewhat greater measure than before, and withal, to look more deeply into the motives of men no longer living, came to me, as to many persons, as a refreshing breeze.

His misgivings as to the shortcomings of Freud in the sphere of ethics is admirably set forth in his recently published book on "Human Emotion," which summarizes his views on the ethical problem, while acknowledging his full debt to the psychanalytical method of approach.

Believing as he did in the positive contribution of Freudian psychology, he was often distressed by what he regarded as illiberal criticism and misinterpretation; but in spite of occasional outbursts of indignation, unusual in him, he remained tolerant, receptive and generous-minded to the end. His convictions were strong, but he was always willing to hear and profit by the opinions of others, and he combined in unusual degree the capacity for independent thought and sympathetic understanding of others' point of view. He was never dogmatic.

He was the last survivor of the original group of seven physicians who were instrumental in founding the American Neurological Association, which held its first meeting in New York, June 2, 1875. These were, in addition to Dr. Putnam, Drs. William A. Hammond, Roberts Bartholow, Meredith Clymer, J. S. Jewell, E. C. Seguin and T. M. B. Cross. During his forty-three years of membership, Dr. Putnam was a faithful attendant at the meetings of the society, and his name appears many times during these years as a contributor and as a kindly participant in the discussions. His record in this respect perhaps does not equal that of his active contemporary and friend, Dr. Mills, but still stands out conspicuously as a characteristic example of his loyalty and devotion to any organization of which he was a member. He served as president of the association in 1888.

His society membership was large, among them being the American Academy of Arts and Sciences and the Association of American Physicians. Before the latter society a number of his earlier papers were read, but during the later years he was inclined rather to contribute to the meetings of the Psychopathological and Psycho-analytical Association, where he might expect a more sympathetic audience. He was peculiarly sensitive to criticism which savored in the least of flippancy; he was, in fact, a poor controversialist and was devoid of a certain sense of humor, which had he possessed it, might have helped him over many hard places. He had many friends but few intimates; he was, however, essentially a social person and keenly enjoyed contact with his fellows, as many of the older men will attest. In a notice of Dr. Weir Mitchell, whom he greatly admired, he speaks feelingly and modestly of their meeting when he was still a young man "at the foot of the ladder," and of Dr. Mitchell's kindness to him on that occasion, "dining at his table while still in the greenness of relative youth and eating there the fattened terrapin." Speaking of Dr. Mitchell's vigor up to the end of life, he writes of him what might well be applied also to himself:

> So predominantly characteristic was this endurance, until the last, of his vivacity, his eagerness and his activity, that one saw at once in him a man of the type made deathless by Tennyson's "Ulysses," longing, even in his closing years, to re-live the days that had been so full of joys and combats among his peers and seeking to the very last new friendships and new worlds.

With Sir William Osler he stood on terms of intimate friendship, a feeling which was cordially reciprocated. He counted among his closer friends Dr. Henry P. Bowditch, Dr. Oliver F. Wadsworth, and especially Prof. William James, with whose habit of thought he had much in common. But in greater degree than with most men he reserved his more personal feelings of intimacy for his immediate family and for the wide circle of his relatives with whom there existed a peculiarly happy sympathy and companionship. He married rather late in life Miss Marian Cabot, a daughter of Francis Cabot of Brookline and Boston, and Louisa Higginson Cabot. His wife, two daughters, one of whom is a student of medicine, and a son survive him, the latter a physician.

It is difficult and perhaps unnecessary to speak at length of his wider life in the community in which he and his family always occupied a position of unique respect. His kindness and consideration for the poor and afflicted knew no bounds; it was natural for him to make personal sacrifices for those less fortunate than he, and it was equally natural for him to conceal his well-doing under a cloak of extraordinary modesty and self-abnegation. Active as he was in his professional work, he found time for divers good causes; it was hard for him to refuse any task which he felt it his duty to perform. He was a prominent and active member of the Society of Mental Hygiene, and gave to it much thought and attention. The associated charities for years demanded and received a goodly share of his time. On the inception of the social service movement at the Massachusetts General Hospital, he at once became identified with its activities, was a member of its executive committee, and gave freely of his time and resources to further its interests. Any movement, in fact, which made a human appeal and which offered the slightest opportunity of helping his unfortunate fellowmen enlisted forthwith his warmest sympathies. It may be said that he deserved small credit for this, since his attitude was a distinctly family characteristic, exemplified also in his brother, the late Dr. Charles P. Putnam, and in his sister, now well over 80, who finds her age no barrier to the prosecution of manifold good works. Be that as it may, the fact remains that Dr. Putnam served his community with a disinterestedness which finds few parallels.

Apart from the strictly professional activities which occupied a large share of his attention throughout his long life, he found time for recreations of a simple sort, into which he entered with the same zest that characterized his more serious work. For many years, he maintained a camp in the Adirondacks with his friend Dr. H. P. Bowditch, known familiarly as the Putnam-Bowditch camp, to which he journeyed yearly during the month of September. There he was accustomed to entertain his friends. Were the history of this camp ever to be written, it would reveal the presence from time to time of many notable men who shared and delighted in its cordial hospitality. Dr Putnam also had a summer place at Cotuit on Cape Cod, where his garden and sailing, and his friends divided his attention. Even in his recreations, however, it was hard for him to lay aside the more serious affairs of life. He was apt to have a book in his pocket, and conversation could not long be kept on minor themes. His interest in nature, and in fact in everything animate and inanimate, was extreme; he enjoyed walking particularly, and was quite able to indulge this pleasure with vigor until very shortly before his death; and even then, the warning of his friends and family deterred him rather than his own sense of incapacity.

His attitude toward the war was characteristic. He was wholly unreconciled to its occurrence; it was constantly in his mind, to the detriment of sleep and of personal pleasure; he seemed to feel toward it a certain personal responsibility — that somehow he was in a measure to blame for the evil deeds of his fellowmen. He had small capacity for lightheartedness when such events were possible in a world which he was forever trying to idealize. He was in a sense a dreamer, and always an incorrigible idealist; tolerant as he was of the opinions of others, he found smallmindedness and narrowness of vision impossible to understand. He was sympathetic with human frailty, but not with bigotry or wilful misdoing. His religion was not so much one of profession as of action; he believed deeply in the beneficent ordering of the world and did his share cheerfully toward trying to make it better and toward reconciling its apparent contradictions and inconsistencies. Men are indeed rare in any community who combine in such superlative degree as did Dr. Putnam devotion to family and friends, a sense of civic responsibility and professional attainment. He was, as President Lowell wrote, in officially commemorating his death, "a man of science eminent in his field, a philosopher and a saint."

His death was sudden. While apparently recovering from a severe anginal attack, he was about to welcome his physician, who had been hastily summoned. The effort was too much for his overburdened heart, and he died with hand extended in friendly greeting, symbolic of the graciousness of his life. He had known for a year or more of his precarious condition, but this in no way abated his activities, which seemed rather to increase than diminish as he knew the end to be approaching. The day before his death he had spent in the open with friends at Concord. His unpublished manuscripts give evidence of his unfaltering mental activity up to the very end. It is a commonplace to say that sudden death is to be desired, nor indeed is this always true; but with Dr. Putnam more than with most men, a period of waning intellectual vigor would have been a calamity, and his family and friends were thankful that he was spared this ordeal.

From his earliest years of practice he devoted himself to his chosen field of work with unflagging zeal and industry. It is hard for us of a later generation to realize the difficulties of establishing neurology in hospital and medical school as a recognized and deserving branch of medicine; he was one of a small and earnest group of men who felt that the time had come for an intensive study of the nervous system in this country, and to him and his contemporaries, some of whom happily are still active in our midst, the younger generation owes a distinct and lasting debt of gratitude.

ELMER ERNEST SOUTHARD

1876-1920

Obituary

ELMER ERNEST SOUTHARD, M.D.

The announcement of the death of Dr. Elmer Ernest Southard comes as a great shock to a host of friends and to American neurologists and psychiatrists. He died in New York City, Sunday, February 8, after a two days' illness with pneumonia. He had gone to New York on Sunday, February 1, to attend the annual meeting of the National Committee of Mental Hygiene and to give an address before the New York Neurological Association. Thursday night he became ill, pneumonia and septicemia developed, and he died the following Sunday.

Elmer Ernest Southard was born in Boston, July 28, 1876. He was the son of Martin and Olive Wentworth (Knowles) Southard. His earlier school training was obtained in the public schools of Boston, and his preparation for college at the Boston Latin School. He entered Harvard College in 1893 and received the degree of A.B., magna cum laude, in 1897. He attained final honors in philosophy, and honorable mention in natural history. In his undergraduate period he was deeply interested in philosophy and psychology. He devoted much time to the courses in logic by Professor Royce, and the foundation he developed there appeared in the mental attitude he maintained in his teaching and research activities in his medical career. It gave to him a habit of clear thinking and an unusual charm of expression that made his contributions and ever ready discussions hold his hearers with keen interest. Even during the busy years of his medical life he found time to attend the seminaries of Professor Royce, and a number of his contributions had their origin in some paper he had prepared for a seminary discussion. This interest in logic always kept him interested in classifications and diagnostic schemes, and determined his method of attack on problems of psychopathology.

In college he was famed as a chess player, and was the holder of an intercollegiate chess championship. His interest in the game was maintained throughout his life, and was for him a source of pleasurable relaxation.

The year of his graduation, 1897, he entered the Harvard Medical School, from which he graduated in 1901. He became intern and assistant in the pathologic department of the Boston City Hospital.

There, under Professor Mallory, he was one of a group of young men of unusual spirit, whose later careers have become conspicuous in teaching and research.

It was at this time that Southard's interest seems to have been directed toward neuropathology. Interest in this subject had been much stimulated by the recent contributions of Carl Weigert to the neuroglia; and the more recent work by Mallory led him to become interested in the problems of the neuroglia, a field in which he always was keenly interested and to which he contributed some of his best work.

In the spring of 1901 he went to Europe and worked for some months with Carl Weigert at the Senkenberger Institute in Frankfurt. From there he went to Heidelberg, where he attended the clinics of Kraepelin and worked in the laboratory of Nissl. On his return to Boston in the fall of 1902, he resumed his work in pathology at the City Hospital. He received the degree of A.M. from Harvard College in 1902. His first medical contribution appeared soon after this, being a study with W. R. Brinkerhoff on "Erythroagglutinins in a Cyst Fluid."

In 1904 Southard was appointed instructor in neuropathology in the Harvard Medical School; he held this position until 1906, when he became assistant professor of neuropathology. Too much cannot be said of the excellence of his course in neuropathology. He brought to it ingenious methods of instruction, and with great success builded the foundations for what later became a separate department of neuropathology in the medical school. His methods of instruction were embodied in his "Outlines of Neuropathology," published in 1906.

Following his return from Europe, Southard continued the interest he had developed in psychiatry while at Heidelberg. He was a most welcome and frequent visitor to the nearby State Hospital for the Insane at Danvers. There he took a keen interest in the work of the laboratory and in clinical visits with the staff to the wards. He thus established between the Danvers hospital and the neuropathologic department of the Harvard Medical School a voluntary but close cooperation in neuropathology that continued for many years.

In 1906, while still assistant professor of pathology, Southard was appointed pathologist and assistant physician to the Danvers State Hospital. Here, with abundance of material, he was most active in research. His alert mind found problems for study in a wide variety of subjects. His unusual capacity for stimulating those who came in contact with him led to a marked productivity in laboratory and clinical studies that continued for some time after he left the hospital. In the three years he was at Danvers his name appears as author or co-author of more than twenty-five contributions. The larger number of these were neuropathologic. An epidemic of dysentery in the hospital furnished material for several important papers on the bacteriology of that disorder. It was at this time that he was associated with Dr. F. P. Gay, and for a while he worked on problems of anaphylaxis. The results of this were published in a series of papers appearing in 1908 and 1909.

In 1909, through the generosity of Dr. W. N. Bullard, Harvard Medical School established the Bullard professorship of neuropathology, and Dr. Southard was chosen to fill the chair. In this year he resigned his position at Danvers to become pathologist to the Massachusetts State Board of Insanity. His new field of work gave him supervision of the scientific work in all of the state hospitals for the insane in Massachusetts. He inaugurated a close cooperation in scientific investigations of psychiatric problems that has continued to this day.

For several years he, with others, had been greatly interested in the establishment of a psychopathic hospital in Boston. In 1909 a legislative appropriation made this possible, and from then until its opening in 1912, Southard devoted much energy toward planning for the new organization, of which he was to have charge.

In 1911 he was seriously ill from an infection acquired at a necropsy. To recuperate from this he spent a few months abroad, traveling in England and Switzerland. He returned in good health, and in 1912 took up his work as director of the Boston Psychopathic Hospital. He now had fields of work that provided great possibilities for his unusual talents. In all, his energetic personality found some problems that interested him. His interests had a wide range, but he saw in all that he did a direct relation to the functions of a psychopathic hospital as he conceived it. This conception has been embodied in several of his contributions, and was never lost sight of. It was that "the psychopathic hospital should be a center of scientific investigation into the nature, causes and treatment of insanity." He formulated these purposes in programs, and the systematically compiled reports of the scientific work done under the joint cooperation of the State Commission of Mental Diseases and the Boston Psychopathic Hospital show how well his programs were adhered to.

He was always able to attract workers around him, and his personal influence on these will be one of his greatest gifts to medical progress.

There is one special field of work that will be inseparably connected with his memory. This is the development of psychiatric social work and nursing. He had the conviction that the "range of psychiatric advance should be from a medical to a medicosocial range," and that "the outpatient and social service relations of a psychopathic hospital must be among its most important functions." The splendid psychiatric social service organization at the Boston Psychopathic Hospital will long stand as a model for other institutions. This interest led to his personal initiative in the organization of the School for Psychiatric Social Workers, established at Smith College in 1918.

His broadened clinical interest is seen in the number of contributions of analyses of group clinical material, and his interest in efforts at the correlation of anatomic changes with clinical symptoms. Along this line, his work on dementia praecox and the Waverly researches in the pathology of the feebleminded form what are probably his best known contributions. The Waverly researches were published in monographic form as Memoirs of the American Academy of Arts and Sciences, and in every way are among the most important American contributions to neuropathology.

A bibliography of his contributions published through his regrettably short professional life embraces more than 150 titles. He was the author of two books of importance, one in collaboration with Dr. H. C. Solomon on neurosyphilis, published in 1917, and "Shell Shock and Neuropsychiatry," which has only recently appeared. In both of these his material is presented in case histories, a method of teaching that always appealed to his logical habits of thought.

His interest in prophylaxis of mental disorders and the field of mental hygiene had convinced him of the value of extending methods of psychiatric social work to problems of industries. He had succeeded in securing the cooperation of a number of large industrial organizations in this work, and had begun a work of great possibilities for good.

During the war, Dr. Southard served as major in the Chemical Warfare Division. He entered this at the solicitation of those in charge of this service, who realized his great powers of personal influence and the benefit these would be to the service in matters of morale. Until then he had served as director of the Boston unit of the U. S. Army Neuropsychiatric Training School, and by his personal instruction prepared many reserve officers for neuropsychiatric work in the Army Medical Service.

His breadth of interest is shown in the number and variety of learned associations in which he held membership. These were: the American Academy of Arts and Sciences; the Association of American Physicians; American Medico-Psychological Association, of which he was president in 1919; American Medical Association; American Association for the Advancement of Sciences; American Neurological Association; American Association of Pathologists; Society of Experimental Biology; the Boston Society of Neurology and Psychiatry; National Association for the Study of Epilepsy; American Psychopathological Association; American Genetic Association; Massachusetts Medical Society; New England Psychiatric Society. He was a director of the Eugenics Record Office and a member of the Board of Scientific Directors of the Bedford Hills Laboratory Bureau of Social Hygiene and of the National Committee of Mental Hygiene.

Since the foundation of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, he was a member of its editorial board. He was on the editorial board of the *Journal of Clinical and Laboratory Medicine,* and of the *Psychiatric Bulletin.*

In 1917 he was honored by the degree of Sc.D. from George Washington University.

In 1906 Dr. Southard married Dr. Mabel Fletcher Austin of Boston, lecturer in Wellesley College, who with two sons and a daughter are his survivors.

Southard was a rare personality. He was by type of mind a philosopher. This gave to his psychiatric interests a breadth of view that led him into widely varying fields of medicine and human thought, and largely determined his method of attack on scientific problems. He was a neuropathologist of eminence, whose contributions are notable additions to science. He was a teacher, brilliant and forceful. As an official of the state of Massachusetts, he gave to the public service great energy, and his stimulating leadership and scientific ideals contributed much to elevate the standards in the care of the insane.

His genial humor, his brilliant powers of expression and his broad intellectual interests leave an ineffaceable memory with those who knew him. He was a type of personality whose greatness was peculiarly felt in its stimulating influence on those with whom he came in contact.

His qualities of mind were such as would have made him eminent in whatever field he might have chosen to work. It is for us of the neuropsychiatric field to feel honored that he chose to be of our number.

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Abstracts from Current Literature

THE FUNCTIONAL SIGNIFICANCE AND PRINCIPAL SYNDROME OF THE CEREBELLUM. FREDERICK TILNEY, Neur. Bull. **2**:289 (Aug.) 1919.

To abstract this article is to do violence to it. The facts presented are packed so closely that it is difficult to condense them without marring the work. The object of this digest, therefore, is merely to sketch the salient features and to suggest that only a reading of the original can do justice to it.

The article, which takes up the whole August number of the Bulletin, is divided into three parts. The first part is devoted to "A General View of the Evolutional Significance of the Cerebellum," the second deals with its "Functions" and the third with "The Principal Cerebellar Syndrome and Its Variations." A case of congenital cerebellar agenesia is described to illustrate the last part.

Part I.—The cerebellum is one of the three suprasegmental portions of the brain. The differentiation from the segmental parts of the neuraxis lies in the fact that for the purpose of expansion the gray matter of the cerebellum is on the surface surrounding the white, and secondly, that the connections with peripheral receptors are less direct, permitting less immediate reflex action, but greater degrees of correlation.

The cerebellum is phyletically of great antiquity; an analogous structure is even said to appear among arthropods. It is constant in all vertebrates and its complexity increases in proportion as the range of movements of the animal advances from the simple to the complex.

In cyclostomes the cerebellum is but little developed; it appears as two dorsolateral evaginations of the metencephalon with direct connection with the somatic sensory tracts. There seem to be present forerunners of the Purkinje cells as well as prototypes of cellular elements later found in the dentate nucleus. In selachians (sharks and rays) the cerebellum is more developed. One can distinguish a granular and molecular layer and large cellular elements with histologic characters of the typical Purkinje cells. The organ has extensive connections for the reception of impulses from the muscles, skin, ear and eye and the gustatory sense. In teleosts and ganoids the cerebellum is similar to that of selachians, except that the splanchnic sensory nuclei are much larger, due to the greater development of the gustatory organs in the bony fish and ganoids. In amphibia the cerebellum is much smaller because of the more sluggish habits of these animals. The granular, molecular and Purkinje layer, however, are well marked. In reptiles the cerebellum is generally small but its size varies according to the activity of the animal. In birds the central arch is enormously developed and forms the vermis, its size being larger in those that fly than in birds that mainly stick to the ground (ostrich, cassowary).

In mammals the cerebellum reaches its fullest development, the large lateral hemispheres being its most conspicuous feature. But there are four essentials to the mammalian organ, namely: (1) a large and complex central arch—the vermis; (2) enormously expanded lateral evaginations—the cerebellar hemispheres; (3) persistence of the flocculus and paraflocculus in rudimentary form, and (4) the presence of a fully developed dentate nucleus and other medullary nuclei.

The size and complexity of the cerebellum in mammals are in direct proportion to the extent of their motor activities, the primates having by far the most highly developed organ. Among the anthropoid apes those who live an arboreal life show great development of the vermis and upper surface of the hemispheres, while in those that tend to become bipedal, whose forelimbs are freed from the necessity of locomotion and tend more toward skilled coordination, the inferior surface of the hemispheres is greatly developed. This accords with the general conception of cerebellar localization.

"The Generalized Pattern of the Cerebellum in Mammals." A comparative anatomic description of the organ is given, based on the works of Elliot Smith and Bolk Smith, the main point of which is to correlate the different parts of the cerebellum with the functional activity of bilaterally and unilaterally synergic groups of muscles in the head, eyes, trunk and limbs.

Throughout the vertebrate series the cerebellum presents a striking uniformity of internal structure, suggesting a definite specialization of function, the ground plan of which is: (1) an outermost zonal layer; (2) a molecular layer; (3) Purkinje cell layer; (4) external medullary stratum; (5) granular layer; (6) internal medullary stratum. The medullary nuclei consist of the nucleus globosus, lateralis (later dentatus), emboliformis and nuclei tecti or fastigii.

In the primitive cerebellum there are connections with several somatic sensory nerves, notably the fifth, with the vagus, the gustatory apparatus, with somesthetic tracts—the optic and auditory (including the vestibular). The main connections are by way of the brachium anterius and posterius, corresponding to the superior and inferior peduncles in higher vertebrates. In mammals the cerebellar connections are made by: (1) the inferior peduncle which contains the dorsal spinocerebellar tract, secondary connections with the columns of Goll and Burdach and olivocerebellar tract; (2) the juxtarestiform body with the Deiterocerebellar tract and secondary connections with the substantia gelatinosa (trigeminal to vermis); (3) the superior peduncle for dentatomesencephalic (red nucleus) fibers and dentatodiencephalic (thalamus) fibers, also possibly for fibers from the red nucleus to the dentate; (4) the middle peduncle for occipito- temporo- parieto- fronto-pontile fibers to the cerebellum. (5) the ventral spinocerebellar fibers by way of the lateral aspect of the superior peduncle to the vermis.

"Summary of the Evolutionary Significance of the Cerebellum": 1. The cerebellum is a suprasegmental addition to the neuraxis in the interest of coordination. 2. In its primitive form it correlates all the sensory impulses for the purposes of coordination. 3. In the lower vertebrates the main structure is the central arch (vermis) which exerts a bilaterally synergic control over the axial musculature, and the primitive flocculus and paraflocculus permit limited, independent movements of the tail. 4. With the development of the upper and lower extremities there appeared the lateral hemispheres which provided unilaterally synergic control of the individual limbs. 5. As a consequence, the greater the range of independent movements, the greater the cerebellar control. 6. The greatest range of skilled movements being seen in man, he has the most highly developed cerebellum. 7. The vermis still retains control not only of the bilaterally synergic movements of the eyes, face, tongue, jaws, larynx, neck and trunk but also of the extremities which require such control.

Part 2.—The author reviews the clinical and experimental evidence adduced as to the functional significance of the cerebellum. He details the investigation concerning cerebellar localization and the results of experimental stimulation of the cerebellar cortex. Further, the results of Bárány's clinical experimentations, together with clinical evidence, are quoted in support of the view of exact cerebellar localization. Finally, the work of Mills and Weisenburg is critically analyzed as to the significance of the cerebellum in synergic control.

Based on a study (to be reported elsewhere) made with the kymograph and slow moving picture the conclusion is reached that synergia depends on two factors: First, the establishment and maintenance of synergic muscular units, and, second, the establishment and maintenance of coordination between the synergic units in the performance of complex acts. By a synergic unit the author means a group of antagonistic muscles which, however, act together in the execution of a movement. For instance, in flexion of a part there is not contraction alone of the flexors and relaxation of the extensors, but both flexors and extensors contract together—the former more vigorously, acting as the dominant factor in the movement, and the latter less vigorously, acting as the check element. When there is a dissociation in the synergic contractions of the opposed groups there is overaction of one group, because of the absence of the check element, and asynergia or ataxia results.

Simple synergia is the action as a unit of an agonist and its antagonist. Integrative synergia is the coordination of two or more synergic units for the execution of a complex act. Synergic units have kinetic and akinetic phases, that is, periods of contraction which, in the first, represent production of motion and in the second a fixed position. For the maintenance of the proper synergia both the force and the extent must be accurately gaged.

Integrative asynergia is divided into several types, namely: *appendicular,* involving the extremities; axial, involving the axial musculature; trunkal and axio-appendicular.

An interesting conception of the author is contained in the chapter on "Synergia in the Light of Cerebellar Histology." He suggests that the "climbing fibers" of the single Purkinje cells have to do with the regulation of simple synergia units; the "basket fibers" make possible the grouping of many Purkinje cells, and the "granule cells" correlate a vast number of them. For this reason and also because the primary function of the cerebellum is to exert synergic control for the regulation of muscular patterns, localization in it, while definite, is not so discrete as in the cerebrum.

"Summary Concerning the Functions of the Cerebellum":

1. Synergia is the fundamental cerebellar function.
2. The entire musculature of the body is divided into synergic units.
3. Simple synergia depends on the proper correlation of simple synergic units.
4. Integrative synergia depends on the proper correlation of many groups of synergic units.
5. Asynergia or incoordination is the essential cerebellar symptom.
6. Asthenia and atonia are symptoms secondary to asynergia.
7. Synergic control of the cerebellum is ipsilateral for muscles of the same side of the body, but bilateral for axial and trunkal muscles.
8. Synergic control of appendicular muscles is both bilaterally dependent and unilaterally independent, the latter more prominent in the arms.
9. Cerebellar localization is more diffuse than cortical because the former regulates entire motor patterns of an act while the latter controls its purpose.

10. The superior and inferior vermis control the synergic action of the axial and trunkal muscles.

11. The superior vermis controls the movements of the eyes, neck, jaws, face, tongue, larynx and pharynx.

12. The inferior vermis controls the muscles of the trunk and the bilaterally dependent movements of the extremities, e. g., walking.

13. The cerebellar hemispheres control the synergic action of the extremities in bilaterally independent acts, e. g., movements of one arm alone.

14. There may be separate areas for adduction, abduction, etc., but they are to be regarded as parts of more comprehensive regions controlling the movements of extremities.

Part 3.—The case illustrating the principal cerebellar syndrome is that of a man aged 41, who had showed marked disturbances of equilibrium since childhood. There was nothing in his past or family history to account for his condition. Neurologic examination showed: All deep and superficial reflexes were normal except for markedly pendulous knee jerks. Volitional control was normal but there was impairment of skilled acts due to asynergia. The muscle tonus was normal. His gait was reeling, there being no coordination between the movements of his legs and body. There was marked decomposition of movement in walking—asynergia major of Babinski. He showed equal difficulty in standing either with eyes open or closed. Nonequilibratory incoordination was very marked; past-pointing was spontaneous; dysmetria was shown in grasping, reversal of hands and heel to knee tests; adiadokocinesis; the rebound phenomenon of Holmes and tremor of action were found. Nystagmus was irregular, jerky and constant; the head oscillated on turning; speech was scanning, interrupted or explosive. All the other cranials were normal and the rest of the neurologic, systemic and laboratory examinations were negative. Mental status was normal.

In his interpretation and anatomic analysis, the author explains the motor disturbances by the failure of the proper actions of the synergic units of the body. The pendulous knee jerk is caused by the inadequate relation between the dominant and check elements in the synergic unit for flexion and extension of the leg. The disturbances in equilibratory synergic control, both simple and integrative, were shown in the station and gait The disturbances in the nonequilibratory movements, in the cephalogyric movements, in articulation and mastication, as well as the nystagmus and the tremor, were due to dissociation in the synergic units. The fact that the idiodynamic, tonic, volitional and automatic associative control were normal and that sensation was intact absolved the rest of the nervous system and incriminated the cerebellum as the cause of the ataxia.

The clinical picture is recognized as the syndrome of cerebellar agenesia or the cerebellar syndrome of Nonne. Primary progressive cerebellar degeneration of Holmes (a familial disease) and olivo-ponto-cerebellar atrophy of Thomas are mentioned as likewise showing the cerebellar syndrome. The author details all the signs and symptoms which accompany extracerebellar conditions in order to differentiate the pure cerebellar syndrome from them. He finally summarizes the clinical features of the syndrome. He insists that atonia and asthenia are not the result of cerebellar disease but are the consequences of the involvement of adjacent structures, which is rather opposed to the views of Gordon Holmes as expressed in his recent studies on the cerebellum. The author also demands further observation before he would accept catalepsy as a symptom due to a defect of cerebellar function.

It is rather doubtful whether this summary review of the very extensive paper does justice to it, particularly to the original contribution which the author has made with his conception of the synergic unit. The great value of the paper lies in the detailed anatomic, physiologic and clinical descriptions and in their successful correlation. One is greatly impressed by the scholarship shown in the article, and any one who values the fact that the only way to understand clinical neurology is to stress anatomy and physiology, cannot fail to appreciate the paper. Particularly valuable is the comparative anatomic study of the cerebellum, as it helps us to understand the functional significance of the organ from a phylogenetic point of view.

Wechsler, New York.

ACUTE IDIOPATHIC OR FULMINATING PSYCHOSIS. C. Ladame, Arch. Suisses Neurol. et d. Psychiat. **5**:3, 1919.

Few mental affections have as intricate a history as acute delirium; up to the present time, writers are far from agreement on this subject and the most divergent opinions are held, which are often apparently equally well founded. The writer prefers the designation of acute idiopathic or fulminating psychosis as describing more appropriately the clinical impression of this fatally terminating malady. A host of appellations have appeared in medical literature to designate related phenomena. These may be divided into two types: first, acute symptomatic psychoses, or acute delirium, which is nothing more than a syndrome appearing in a large variety of mental and physical disorders; second, acute idiopathic psychosis, a clinical entity, which progresses in a fulminating manner to a fatal termination. The histopathology, if not specific, is none the less very characteristic.

The etiology of this disease is unknown; the majority of patients reveal some hereditary taint. Of eight cases of acute idiopathic psychosis observed at Bel-Air, all were females; the mortality was 100 per cent. These patients had reached full maturity, the disease occurring between the ages of 25 and 45. The affection is rare. Its course is almost precisely the same in all cases and is somewhat as follows: there is often a slight prodromal period of fatigue and loss of appetite, but the psychosis, itself, begins brusquely and immediately indicates an affliction of greatest gravity. At the end of the first day, the patient reveals all of the clinical characteristics; exitus occurs almost regularly in the first week, rarely in the second, and in a few isolated instances in the third. Cachexia develops with extreme rapidity. The first day is distinguished from the following by the fact that personal consciousness is fully retained; the patient is lucid and presents no difficulty in orientation. However, there is a marked restlessness and anxiety, which continues throughout the course of the disease. Soon the patient becomes confused, the lucid intervals become shorter and shorter and presently marked hallucinations appear. His attention can be held for but a moment. Consciousness is soon replaced permanently by psychic nothingness. Intense psychomotor activity continues, the movements being elementary and incoherent. The patient absolutely refuses liquids and nourishment, spitting them out with an expression of horror. Doutless this sitophobia is based on delusions of poisoning. An extreme fetor is noted about the patient, which possibly furnishes the basis for hallucinations of taste and smell. There is complete insomnia; the urine is scanty and shows slight traces of albumin and deposits of phosphates; there is an abundant acrid perspiration; salivation is copious and the patient expectorates continually early in the disease. Little by little these excretions dry up; the tongue becomes dry, black, and hard; the lips and gums are retracted and bleeding; the skin looks dry and withered. There is an odor sui generis, which permits diagnosis almost at once. Cardiac action is rapid but feeble; often there is some elevation of temperature with antimortem exacerbation, and bronchopneumonia, pneumonia, and pulmonary edema are noted. Intercurrent diseases play a minor rôle as a cause of death.

Although it is difficult to attempt an explanation of the pathogenicity of an affection of which the necropsy is practically mute, the author feels that certain deductions can safely be drawn owing to the fact that these patients succumbed to an extreme cachexia in which a third to one half of the weight of the body is lost within a few days. It is a fact well known and supported by ample observation on other diseases, that such a marked and sudden loss in weight brings about a quantitive disturbance of the water balance of the organism. There is a considerable and generalized lypoidolysis with an intense disintegration of lecithin as well as of cholesterin. It has been demonstrated that the cell membrane has a very high lecithin coefficient and plays the rôle of an osmotic membrane. Granting the disintegration of lecithin, the semipermeable cell membrane loses its integrity; as a result, a serious osmotic disturbance appears in which the intracellular and extracellular equilibrium of liquids is lost. To this purely mechanical factor is added the action on the tissues of liquids laden with noxious substances. The organism is thus subjected to an intense dehydration. Thus, although we are ignorant of the primary cause of the malady, we know better the succession of morbid processes, once the disease has been initiated.

Necropsy reveals a strong meningeal and encephalic hyperemia; the ventricles are almost deprived of liquids; cerebral edema is present, the cortex presenting the picture of a marked état criblé. Nerve cells and fibers show marked accumulation of isolated and confluent droplets staining with Scharlachrot. The same picture is noted in the cerebellum. There is a marked chromatolysis and karyolysis, cellular sclerosis, and an intense neuronophagia. With toluidin blue, a beautiful metachromasia is noted in the nervous and neurogliar elements. The neuroglia cells are often completely filled with fatty droplets. The blood vessels are engorged with blood to their finest ramifications; a number of them are obstructed with hyalin thrombi. The endothelial cells are distended with lipoid droplets to the point of complete obliteration of the lumen. The perivascular lymphatic spaces are dilated and filled with lymphocytes, mast cells, plasma cells, and Abbauprodukte. The prognosis is bad, exitus occurring early. The writer is of the opinion that many cases diagnosed as acute idiopathic psychosis, which recover, really belong to the type of acute symptomatic delirium. Treatment is purely symptomatic though it would appear that liquid should be administered in large amounts.

Woltman, Rochester, Minn.

THE ENDOCRINE ORIGIN OF MUSCULAR DYSTROPHY. N. W. Janney, S. P. Goodhart, V. I. Isaacson, Arch. Int. Med. **21**:188 (Feb.) 1918.

The authors have investigated the metabolism in nine cases of muscular dystrophy with the view of determining whether that disease "may originate as the result of dysfunction of the endocrine organs." The cases studied were typical dystrophies; practically all showed evidences of trophic changes in the nails, skin, hair, etc.; some showed peculiar hair distribution; all showed rarefaction of the skull and long bones. Most of the patients were young, only two were adults. Of the group, eight were males.

Careful determinations were made of the creatinin-creatin metabolism, total nitrogen, blood sugar and blood sugar tolerance curves. The calcium and magnesium metabolism was studied in only one case. The diet was creatin-creatinin free. Daily protocols were made of the urinary findings in twenty-four hour specimens.

A summary of the experimental results showed: (1) marked decrease in the preformed creatinin in the urine; (2) abnormal presence of creatin in the urine; (3) low values of creatinin in the blood; (4) normal amount of creatin in the blood; (5) hypoglycemia; (6) delayed glucose utilization (hourly blood sugar curve). Five out of nine cases showed glycosuria after ingestion of sugar in relatively small quantities (exact weights not given). Total urinary nitrogen was low in all cases, but blood urea nitrogen seemed to be normal in five cases. In one case the calcium and magnesium output was low, indicating a probable retention.

As to the clinicopathologic evidence of the endocrine nature of muscular dystrophies, the authors point to the marked bony changes found in patients suffering from those diseases and try to correlate the studies of others made in cases of endocrine disturbance. Of other changes referable to endocrine disorders the authors mention dryness and brittleness of hair; pigmentations, hypertrichosis, peculiar fat distributions and underdeveloped genitalia as having occurred in their series of cases.

The creatin increase in the urine is shown to be similar to that found in starvation, particularly carbohydrate starvation, and the authors maintain that the dystrophy owes its origin to the hypoglycemia which is found. The presence of hypoglycemia in myxedema, cretinism, dyspituitarism and Addison's disease is mentioned to show by analogy the possible endocrine origin of the dystrophies. To show that the hypoglycemia is not due to disuse atrophy of muscles, the authors studied the blood sugar in cases of chronic arthritis with muscular atrophy and found it normal. Experimental removal of ductless glands demonstrated a hypoglycemia.

The authors explain the muscular atrophy on the supposition that it is due to lack of carbohydrates and point to the deposition of fat in the muscles and elsewhere as a sign of hypoglycemia.

Concerning the question as to what endocrine glands are involved in muscular dystrophy the authors review the case reports of numerous other observers, and remark on the want of uniformity of the conclusions reached. Some cases gave symptoms referable to the pituitary, others to the suprarenals, some to the thyroid, etc. The pineal may possibly be a cause in some cases. In the authors' series the presence of acromegaly in one case and possible pineal shadows in two others may point to the two special glands. However, the authors look on the dystrophies more in the light of a syndrome which is the result not alone of disturbance in one gland but in several endocrine organs separately or coincidentally affected.

The study undertaken by the authors is extremely valuable. The evidence, indeed, is not altogether conclusive, but is striking and suggestive, and as the authors well point out, much further evidence may be adduced by scientific therapy. Unfortunately, most of the present day endocrine therapy is of the hit-or-miss variety, possibly because of ill-understood symptomatology and unbridled speculation. All the more valuable, then, is the exact scientific investigation furnished by the authors. They have made a distinct contribution to our knowledge of the metabolism of dystrophies.

Wechsler, New York.

MODIFICATIONS OF THE FINGERNAILS IN PERIPHERAL NERVE LESIONS DUE TO GUNSHOT WOUNDS. Lieut. Guiseppe Carlo Riquier, Assistant in the clinic at the Royal University of Sassari, Riv. di patol. **24**:90, 1919.

Changes in the ungual tissues have not been the object of particular study in peripheral nerve wounds during the war. Examination of many cases has convinced the author that such changes do occur and have a marked diagnostic and prognostic value. Increase in nail growth normally occurs to the extent of about 0.1 mm. a day and would equal about 3 mm. a month. Injuries, particularly of the median and the ulnar nerves, will show either an increase or diminution of growth in some or all of the finger nails. He controlled his studies by cutting all the nails at the same level and by painting them with nitrate of silver and measuring daily the nonstained growth which appeared. He found that in the syndromes of irritation of the median nerve more particularly but also of the ulnar, the nails increase in growth rapidly, even in the so-called cases of causalgia although in this condition all the nails were affected. In lesions showing complete interruption of the nerve the growth of nails was greatly diminished. There were also changes in the color of the nails, opacity, size of the lunula, the striations and changes in the lateral and anteroposterior curves. The author agrees that these lesions are also found after infections of the extremities, injury to vessels and even in lesions of the central nervous system. Changes in color, striations and in the curvature of the nails were found in one case of left radial nerve injury. The author feels, however, that these small nail signs in these cases are due to disuse which influences particularly the curvature of the nail because of the atrophy which occurs in the tips of the fingers which build up and give shape and curve to the nails. As a sign of specific irritative lesion of the median and the ulnar nerves the mere change in the curve of the nail is not important unless it assumes a grotesque form in which case a real ungual hypertrophy is characteristic of median nerve injuries. Here, however, one notes also great rapidity of growth with many longitudinal and transverse sulci. In destructive lesions one never sees marked changes in the curvature of the nails. Indentations and depressions with opacity, variations of color, etc., are invariably pathologic signs of irritative or partial lesions. Another important pathologic sign is the presence of a sharp line between the proximal and distal lamina of the nail which sharply delineates the normal proximal lamina from the changed distal lamina which will show various degrees of change in color, shape and size. This may be indicative of a lesion in both the median and ulnar nerves. The author has been able to show in some cases that changes of this type occurring in the last three fingers were more apt to occur with ulnar lesions. In lesions of the sciatic and internal popliteal, disturbances of the growth and morphology of the nails were also encountered, but their study is made difficult by the fact that variations in the type of normal nails of the toes is great, due to interference and modification caused by footgear.

Osnato, New York.

THE EXPERIMENT IN OCCUPATIONAL THERAPY AT BASE HOSPITAL 117, A. E. F. Sidney I. Schwab, Ment. Hygiene **3**:580 (Oct.) 1919.

Schwab presents an interesting account of the experiment in occupational therapy at Base Hospital 117, the A. E. F. war neuroses treatment center. The problem was to return to the firing line, in a relatively short time, as large a proportion as possible of organically sound but functionally defective soldiers. That occupational therapy played a most important part in the healing of these "bloodless wounds" is clearly indicated by the fact that in 85 per cent. of the 3,000 patients who passed through the hospital, occupations of various kinds were utilized as therapeutic agents. The list included stone-breaking, road-building, wood-chopping, tilling, carpentry, tinning, printing, weaving, painting, sketching, wood-carving, etc. The philosophy of work applied to the neuroses, as Schwab sees it, lies in the fact that both the physiologic and psychologic needs of the patients are met and adequately supplied. For instance, even such a simple matter as stone-breaking, exerts a beneficial mechanical effect on the excessive muscular activity which is a concomitant of ataxia and tremors and by the coordinated movements involved favors a tonal balance between antagonistic muscle groups. From the more strictly mental point of view, the patient is impressed by the demonstration of the fact that his illness must be evanescent, since the muscles affected by the physical defect (tremor, palsy, etc.) are capable of performing coordinated movements and are responsive to motor control. Furthermore, work and particularly the more artistic forms of endeavor, supplied an outlet for the emotional hypertension which exists. The author rightly stresses the necessity of regarding occupation as a medicine, which must be individually prescribed by the physician. Neglect of this factor of individuality means paucity of results and possible harm to the patient. The experiment at La Fauche not only proved the feasibility of work as a valuable form of treatment of the war neuroses in the zone of advance, but was further a distinct forward step in the evolution of the occupational therapy idea, which will bear fruit in civil life.

Strecker, Philadelphia.

ANAPHYLACTIC PHENOMENA IN THE PATHOGENESIS OF CERTAIN EPILEPSIES. P. Pagniez and P. Lieutaud, Presse méd. **27**:693 (Nov. 19) 1919.

Starting with an anaphylactic conception of epilepsy, the writers cite one case which can reasonably be viewed from that standpoint. Certain therapeutic tests were applied, and results obtained which were instructive and suggestive within the limits of the investigation.

Widal (*Soc. méd. d. hôp. de Paris,* Feb. 13, 1914) had established an anaphylactic basis for urticaria: certain foods could give rise to cutaneous, digestive, and respiratory disturbances, preceded by blood-vascular changes in the nature of a crisis ("crise hémoclasique initiale"). Other writers had shown that minimal doses of the noxious substance, ingested some time before the meal which would contain it in quantity, prevented both the crisis and the subsequent symptoms. The recognized importance of alimentary disturbances and dietary regulations in epilepsy is cited, and an attempt is made to demonstrate related anaphylactic reactions.

A classical case of epilepsy, which had shown improvement under some dietetic care, and which was not being treated by bromid or other drugs, was selected for experimentation. Ordinary meals of bread, meat, eggs and vegetables, with a little wine, were not followed by attacks, and examination from hour to hour showed a normal digestive leukocytosis and slight elevation of blood pressure. When 50 gm. of chocolate were added to the meal, it was followed by a series of sharp oscillations in the blood count, dropping to a leukopenia of 3,600, accompanied by some lowering of the blood pressure. This was considered a true crisis ("crise hémoclasique" of Widal), and was followed shortly by one or more convulsive attacks. Several days later, when 0.050 gm. chocolate was administered forty-five minutes before the same type of meal, including 50 gm. chocolate, the blood disturbances and convulsive seizures were absent. Both experiments were repeated several times on the same case, with appropriate controls.

Huddleson, New York.

SEVERE POST-DIPHTHERITIC PARALYSIS FROM WOUND DIPHTHERIA. E. Röper, M.D., Neurol. Centralbl. **38**:450 (July 16) 1919.

The war offered unusual opportunities to study diseases and injuries of the peripheral nervous system, especially severe multiple neuritis with paralysis of all extremities from furunculosis, chilling of the body, grip, etc., with complete recovery. Neuritis following wound diphtheria occurred more frequently than was supposed, because the diphtheritic infection of wounds was often overlooked, unless the neuritis following exhibited characteristics of diphtheritic paralysis.

An officer, aged 25, was wounded in the left hand by a hand grenade April 27, 1918. The wound was small and deep. He had a fever of 39.8 C. on the fourteenth day, smear-like exudate in the wound, swelling of the hand, discharge of fetid pus, and necrotic tissue. During the seventh week the patient developed bilateral paralysis of accommodation. In the eleventh week there were numbness of the tongue, lips, fingers and legs, weakness of the arms and legs, absence of all deep reflexes and tenderness of the sciatic nerves. There was general improvement with normal neurologic findings by May, 1919. No bacteriologic study of the wound was made.

A soldier, aged 27, was wounded by a bullet in the left thigh March 10, 1918. He became very anemic from loss of blood. No injury to the nerve trunks was found. The wound remained clean until May 21, when fever and wound inflammation with foul exudate developed. On November 2, neurologic examination showed muscular weakness, anesthesia in the legs, ataxic gait, weakness of the reflexes in the arms, absence of the knee and Achilles reflexes, and the presence of the pharynx reflex. The eyes were normal. The diphtheritic character of the paralysis suggested a cultural investigation of the wound, which revealed diphtheria bacilli. In both instances, the diphtheritic character of the wound infection was first suggested by the results of a neurologic examination.

Shelden, Rochester, Minn.

THE PRESENT POSITION IN CLINICAL PSYCHOLOGY. William McDougall, J. Ment. Sc. **65**:141 (July) 1919.

An historical account of the theories of the workings of the mind is attempted by Major McDougall. He discusses the sensationism in its combination with associationism which gave us our picture of brain-cells connected to switchboards, and led research workers to neuropathology.

The casting aside of mechanistic psychology is best illustrated by the work of Freud, who has brought two great facts to light: (1) "the impulsive, demoniac, illogical nature of much of human thought and conduct;" (2) the poor representation in consciousness of this impulsion. The over-importance attached to sex by Freud has been partly counteracted by later studies of self-assertion, gregariousness and fear. The author emphasizes that these fundamental impulses, and others, are purposive and that our conduct is to be regarded as determined not only by the events of the past but by ideals of the future. Man "is borne on to his biological ends, for the most part but dimly conscious of those ends and of the mental forces and processes by which he achieves them." The conception of these forces, not distinct from consciousness, but greater than consciousness, is of more value than the vague "unconscious" of Freud and Jung. That the individual mind reacts according to its inherited impulses is a final assumption worth much to medicine because it explains "the uniformity of thinking—revealed by primitive myth and custom" and by the symptoms of certain mental disorders.

The reviewer questions whether mechanistic psychology needs to be hurled so far out of the way of the onrushing "clinical psychology"—certainly the structure of our nervous system is worth some study.

Bond, Philadelphia.

FOREIGN BODIES IN THE CEREBRAL VENTRICLES. G. L. Regard, Presse méd. **27**:645 (Nov. 1) 1919.

The writer in discussing foreign bodies in the cerebral ventricles limits himself to such foreign bodies as bullets or fragments of shells, and does not include foreign bodies of a different category, such as hydatid cyst. His experience has shown that foreign bodies of the type he considers are most frequently found in the lateral ventricles and least frequently in the fourth. He expresses the opinion that they reach the ventricle secondarily, having first lodged in the adjacent brain tissue. In connection with naming over the symptoms which are the usual ones of brain injury, he warns against lumbar puncture in these cases on the ground that it occasions a diffusion of microbes from the site of the foreign body.

The author relies on the roentgen ray for diagnosis. Radiographic examinations indicate the intraventricular site of a foreign body, though that fact is definitely established only if successive roentgen-ray examinations show changes of location of the foreign body dependent on changes in position of the head. A foreign body can be, as it were, "tracked" into the frontal or temporal extensions of the lateral ventricle. A foreign body not displacable by the head movement but "caught" in the middle ventricle, is diagnosed on presumption when its roentgen-ray shadow lies exactly beneath the sagittal suture, is 25 mm. above the zygomatic arch and from 0 to 20 mm. in front of the opening of the auditory canal. Similarly a foreign body, not displacable, owing to its size, is given a presumptive diagnosis of location within the lateral ventricle when it lies within the four classic topographical lines outlining this ventricle.

The writer outlines a two-stage operation for the removal of foreign bodies from the ventricles — the latter part of which is done coincident with radiographic inspection. He gives a good prognosis for such cases if only the complication of infection can be avoided.

Davis, New York

Society Transactions

AMERICAN NEUROLOGICAL ASSOCIATION

Forty-Fifth Annual Meeting, held at Atlantic City, N. J., June 16-18, 1919

(Continued from page 208)

21. CEREBELLAR DYSSYNERGIA WITH SPECIAL METHODS FOR ITS STUDY AND ANALYSIS. Adrian V. S. Lambert, M.D., and Frederick Tilney, M.D., New York.

Illustrated by novographic motion pictures, and kymographic tracings.

DISCUSSION

Dr. L. Pierce Clark, New York: This extraordinary and interesting method of case study is certainly delightful both clinically and experimentally. I should like to ask Dr. Tilney whether it has been extended to any other type of motor disorder, for instance, myoclonia and epilepsy. I would like to voice my regret that when I studied the convulsive phenomena of epilepsy in 1898 I searched everywhere to find a machine to produce the same thing that Dr. Tilney has shown in such an excellent manner. What was particularly needed there was to take the rapid discharge and muscle movement phenomena of epilepsy and bring them down to slow movements so that one could study the initial foci in the jacksonian type. Since then the moving picture has been developed. This means of getting at the segmental movements and bringing them in for careful study is of enormous importance as it concerns the whole convulsive phenomenon of epilepsy. The teaching value also is considerable as it will give medical students a more definite knowledge of the motor phenomena in all convulsions. I would like to ask Dr. Tilney if in any of his cerebelloid cases instruction would modify this asynergic behavior. The teaching of graceful movements which can be used all over the field of disease phenomena in the selection of better methods of motor coordination, could as well be applied to normal children in schools.

Dr. Frederick Tilney, New York: I will say just a word in answering Dr. Clark, with reference to applying this method of study to myatonia and locomotor ataxia. It is an expensive undertaking and the pictures of the cerebellar cases cost several thousand dollars. We spent a large sum of money in getting a limited amount of film. Photographing the epilepsies would be difficult and expensive. As to the education of patients with asynergic movements, that has so far not been practicable, but perhaps later on something of that sort will be reported.

22. CONGENITAL FACIAL PARALYSIS. Frank R. Fry, M.D., and Michael Kasak, M.D., St. Louis.

This article appeared in the December number of the Archives of Neurology and Psychiatry, page 638. No discussion.

23. HEREDITARY OCCURRENCE OF HYPOTHYROIDISM WITH DYSTROPHIES OF THE NAILS AND HAIR. Dr. Albert M. Barrett, Ann Arbor, Mich.

This article appeared in the December number of the Archives of Neurology and Psychiatry, page 628.

DISCUSSION

Dr. Beverley R. Tucker, Richmond, Va.: Dr. Barrett has opened an interesting field in the history of the endocrine disturbances. I recall the case of a family in which the father and mother, both absolutely normal, had six children who were all normal except one and she was distinctly a cretin. When she grew up she married an alcoholic and they had seven children, and of these seven children every child suffered from convulsions. One had one convulsion, two had such frequent convulsions that they were sent to an epileptic colony in Virginia. I was more or less fortunate in being able to get roentgenograms of the pituitary of five; three showed no change in the sella, two showed distinct hypopituitary changes and they were put on pituitary extract. One improved to a fair degree, and the other has not had a convulsive attack for over two years. None of them showed any sign of hypothyroidism clinically though the mother was distinctly hypothyroid, except possibly the youngest child who was 2½ years old the last time I saw it. As to the nails and hair, I am afraid to venture discussion on that because I have not my notes with me. The point I would like to emphasize in these endocrine disturbances is that in the succeeding generation we are likely to get not only the ductless gland disturbance of the parent but other ductless gland disturbances and various forms of feeblemindedness, and we cannot predict the absolute relation. It is interesting to note that the father in the case just recited was an alcoholic.

THE PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 21, 1919

J. Hendrie Lloyd, M.D., *President*

AN UNUSUAL SYMPTOM GROUP FOLLOWING INFLUENZA. Presented by Dr. Benjamin Weiss.

An Italian, aged 30, a stone mason, married, whose wife had had three children and no miscarriages, denied venereal infection, and the family history was negative. He was well until October, 1918, when he had a severe attack of influenza, and was confined to bed for a period of two weeks.

In January, 1919, the man began to have difficulty in walking. He stumbled and had poor control of the movements of his legs. His gait gradually became worse and he required a cane in getting about. He also noted that his vision became less acute, more especially that of the right eye. He had had severe occipital headache for several days without vomiting.

Physical Examination.—The gait was staggering, with a tendency to fall laterally, usually to the left. The incoordination became much more marked when he walked with his eyes closed. The station with the eyes open was poor; there was a tendency to fall either to the left or right, or backward. With his eyes closed his equilibrium was greatly disturbed. The eyes were prominent but had been so since childhood. The pupils were equal and round. The right pupil reacted sluggishly to light, but reacted well to accommodation. On extreme lateral rotation nystagmoid movements were noted. No ocular palsies were apparent. The eye ground examination revealed secondary optic atrophy in the right eye and a choked disk in the left.

The upper extremities showed no loss of muscular power. There was some incoordination on the right in the finger to nose test. His penmanship was poor. Adiadokocinesis was present on the right and absent on the left. The reflexes were normal. The lower extremities showed good muscular power in both legs. In the heel to knee test there was some ataxia in the right leg, but none in the left. Ankle clonus and Babinski's sign were absent on both sides. The patellar reflexes were slightly exaggerated. The sphincters were uninvolved and there were no sensory disturbances. The memory was unaffected. The skiagraph of the head showed no evidence of any lesion; examination of the ears revealed no pathologic changes. The blood and spinal fluid examinations were negative.

FRIEDREICH'S ATAXIA IN TWO COLORED BOYS—BROTHERS.

Presented by Dr. J. Hendrie Lloyd.

Dr. Lloyd said his cases were of interest, not only because they were quite typical, but also because they occurred in colored boys. Friedreich's ataxia in the negro was probably rare; at least, Dr. Lloyd did not recall having seen an instance of it before. The patients were full blooded negroes; and for their ages they were fairly well developed and intelligent. The father, aged 39, and the mother aged 35, were both apparently healthy. The mother and one daughter gave negative reactions to the Wassermann test of the blood. The blood could not be secured from the father; but for the question of possible congenital syphilis the test of the mother and daughter's blood was more important. Both patients gave negative reactions from the blood and spinal fluid. Although there was not much ground for supposing that congenital syphilis was a cause of Friedreich's ataxia, it was thought desirable to test this question as fully as possible. The mother had had five children; the eldest, a daughter aged 14, is living and well; two sons, the patient and another son who had convulsions and died at the age of 3; and a daughter, aged 3, who is living and well. There was one miscarriage at seven months. Otherwise the family history was unimportant. There had been no ataxia in collateral branches so far as could be ascertained.

I. C., the elder boy, aged 11, was born by normal labor. He learned to talk and walk at the usual age. He went to school and did well. He had convulsions at the age of 1, and some of the usual diseases of childhood. The present trouble began, when he was 8 years old, with awkwardness in the leg movements, the patient falling to his knees when attempting to walk. He had a marked scoliosis. The gait was ataxic, the feet were placed far apart and the boy swayed violently in walking, which he did with difficulty, requiring assistance. It was not the gait of locomotor ataxia. The station was very unsteady, and was increased on closing the eyes. The finger to nose test and the heel to knee test showed ataxia; there was marked adiadokocinesis. The knee jerks and Achilles' jerks were absent, and there was no Babinski reflex on either side. The abdominal and cremasteric reflexes were present. Sensation to touch, pain, heat and cold was normal, but there was some loss of sense to a tuning fork placed on the tibia, radius and ulna. No shortening of the plantar arch, or hammer toe, which is described as seen in some cases of this disease, was present, but there was marked flat foot, with slight eversion. No loss of sphincter control; no muscular atrophy; no subjective pain; no girdle sense were found. The speech was slightly hesitating or staccato, not drawling. The eye condition is noted below.

M. C., the younger boy, aged 9, born of normal labor, talked and walked at the usual age, went to school and was considered very bright. He had convulsions at the age of 2 and the usual diseases of childhood. The present trouble was first noticed when he was 6 years old, and was recovering from an attack of typhoid fever. Dr. Lloyd said that the boy's gait and most of his other symptoms were so similar to those of his brother that it was hardly necessary to recapitulate them in detail. There was the same gait, with swaying movements, and unsteady station, which however, was not increased on closing the eyes. The deep reflexes were abolished; there was no pain and no loss of sensation in any of its modes, except for the tuning fork, was detected. There were, however, no deviation of the spine, and no muscular atrophy or loss of sphincter control. The speech was slightly hesitating.

Dr. Shannon reported concerning the condition of the eyes: In the elder boy the vision in both eyes was normal. The pupils were equal. The reaction to light, direct and indirect, was sluggish. Accommodation and convergence reaction was prompt. The iris showed a curious lack of pigmentation in the upper nasal quadrant of the right eye, and in the upper nasal and lower temporal quadrants of the left eye, suggesting iritic atrophy at these points. On distant fixation the left eye showed a tendency to rotate outward, although on close fixation fair convergence was obtained, indicating a condition of exophoria. This measured with the Maddox rod about 12 degrees. The characteristic ataxic nystagmus was not elicited. In the right eye the media were clear; the disk discolored and somewhat atrophic; the margins clearly outlined; vessels moderately contracted; macula normal; no changes in the periphery of the fundus. In the left eye the conditions were similar.

In the younger boy, the vision was about normal. The pupils were equal, and reacted sluggishly to light, but promptly to accommodation. the irises were normal. The characteristic nystagmus, sometimes called pseudonystagmus or ataxic nystagmus, was observed when the eyes were fixed on a moving object, especially in the extremes of rotation. In the right eye the disk was discolored and atrophic and the vessels contracted. In the region of the macula a leaden-hued area, about the size of the disk, was covered by a sprinkling of small round or triangular coal black pigment patches. In the extreme nasal field additional pigment patches were seen, and isolated patches of pigment were seen in various parts of the fundus. The left eye revealed the same condition.

It was noteworthy that neither speech defects nor nystagmus were very well established in these cases, but these defects are sometimes rather late in appearing even in typical cases of Friedreich's ataxia. The appearance of slight optic atrophy was also noteworthy. Several cases have been reported in which the light reflex was lost. Gowers thought this an evidence of syphilis. It may be an evidence that the case was one of juvenile tabes.

FRIEDREICH'S ATAXIA IN MIDDLE AGED MEN—TWINS. Presented by Dr. George Wilson.

Dr. Wilson said that the cases which he desired to present were interesting because they were atypical and occurred in twins.

Case 1.—The elder of the pair was admitted to the Philadelphia General Hospital in the service of Dr. Spiller, Sept. 25, 1919. The man said that he was in good health until he was 33 years of age, twelve years ago. At that time he noticed that he had difficulty in walking; this infirmity gradually increased, but was never so severe that he could not walk. A few months after the initial symptom the patient noticed that his speech was becoming "thick." As far as the man was concerned, he had no other symptoms. In addition to his twin, reported below, he had two brothers and one sister who were living and normal. The patient had drunk moderately. He denied syphilis but had had gonorrhea.

Physical Examination.—The man was a well nourished Jew. He was cranky, and did not adapt himself readily to his surroundings. Romberg's sign was present, and the gait was an ataxic, rolling one. Marked clumsiness and ataxia in the hands were brought out when the man dressed and undressed himself. Adiadokocinesis was present on both sides, and a slight intention tremor developed in the finger to nose tests. The irises were brown, the pupils were equal, centric and reacted well to light and in convergence. Horizontal nystagmus was present. There was no exaphthalmos or hemianopsia. Dr. Shannon reported that the optic nerves were normal. The cranial nerves were normal. The speech was thick and slurred.

The patellar and achilles' reflexes were present and lively. The biceps and triceps reflexes were present, but depressed. The cremasteric and abdominal reflexes were present; plantar stimulation produced flexion of the toes on both sides. Pain sense, heat and cold were normal throughout. The sense of position was affected in the toes of both feet. There was no paralysis, no muscular atrophy or hypertrophy, no deformities of the spine or feet and no cerebellar catalepsy. The blood Wassermann test was negative; the patient refused to permit a lumbar puncture.

Case 2.—The second patient was a replica of his brother. The story of his illness and examination was practically the same as the record given above. The second patient was as affable as his brother was disagreeable. He had had both gonorrhea and a chancre although the blood and cerebrospinal fluid were normal. The patellar reflexes in the younger of the twins were distinctly exaggerated without the presence of ankle clonus or Babinski's sign.

Dr. Wilson said that the cases which he had presented were of an unusual type. The symptoms were late in appearing and an analysis of the histories showed many atypical findings. Though the patellar reflexes were exaggerated, this sometimes occurred in Friedreich's ataxia together with Babinski's sign because the pyramidal tract might be degenerated. The deformities of the feet, toes and spine did not always occur and were absent in these cases. Dr. Wilson observed that the cases of Friedreich's ataxia which were presented to the society demonstrated that Jendrassik was correct when he said that the family diseases of the nervous system blended almost inseparably one into the other and that the symptoms were constantly overlapping. Jendrassik also said that a family disease when present in a family might be, and often was, atypical but that all of the members affected were involved in the same manner.

TWO SISTERS WITH FRIEDREICH'S ATAXIA. Presented by Dr. J. W. McConnell.

A. H., aged 22, whose walking had been bad since she was 14, was for a time improved by using a prescribed shoe; but of late she had been stumbling with unusual frequency, even falling without apparent cause

Examination showed abolition of the reflexes in the upper extremities with a very insignificant atrophy of the left hyperthenar eminences with distinct loss of power in the lower limbs and almost complete inability to elevate the toes and foot. The reflexes in the lower extremities were completely lost, not reinforcible; both feet were instances of moderate talipes cavus, and she had the distinct deformity of the toe which is seen in Friedreich's disease. She showed no cranial nerve conditions, no speech defect, no disturbance of vesical or rectal sphincters, no spinal curvature. She had no ankle clonus or Babinski sign. There was no tenderness over the nerve trunks nor were the nerves palpable. She had no sensory disturbance. The sister of this girl, three years younger, presented a similar foot deformity with lost reflexes in the lower limbs.

Her mother had had difficulty in walking, with a deformity of the foot and was for many years previous to her death quite invalided. The maternal grandfather also had had a foot deformity, but so far as could be learned he had had no paralysis or disturbance of locomotion.

Both these cases were regarded as probable instances of Friedreich's ataxia.

DISCUSSION ON FRIEDREICH'S ATAXIA

Dr. W. G. Spiller stated that the three families presented three different types of Friedreich's ataxia. He thought Dr. Lloyd's cases were typical. The twin brothers shown by Dr. Wilson belonged more to Marie's hereditary cerebellar ataxia type, especially in the age at which the symptoms developed, and in the preservation of the patellar reflexes in one and the exaggeration of these reflexes in the other. The girls presented by Dr. McConnell showed the earliest form of Friedreich's ataxia. They had the distinct Friedreich toe, loss of tendon reflexes, ataxia and nystagmus.

Dr. J. H. Lloyd brought forward the question of syphilis as a cause of Friedreich's ataxia. Dr. Spiller said it would be strange that syphilis should affect two members of a family in so similar a degree as in the cases presented. Two cases in each of the three families were much alike, and yet all three types were different. Syphilis in attacking a family so as to produce disease of the nervous system is likely to produce different manifestations —for instance, in one tabes, in another paresis. When husband and wife have contracted syphilis, probably from the same source, the forms of the syphilitic disease need not always be the same.

The optic atrophy and absence of light reflex in Dr. Lloyd's cases occur in the form of the disease described by Marie, which probably should be considered as a part of Friedreich's ataxia.

Friedreich's ataxia is usually regarded as a rare disease, and it was an extraordinary coincidence that three different families showing three different types of the disease should be presented at one meeting. As for being rare in the negro, it follows that it is likely to be so when it is rare in the white race.

From the pathologic standpoint there is much to be said of the relation to one another of several forms of family disease of the nervous system. Family spastic paralysis has pathologic findings resembling those of Friedreich's ataxia. It has been shown that the family form of optic atrophy may be associated with symptoms of posterolateral sclerosis.

Dr. Charles K. Mills said he had nothing in particular to add, except perhaps to call attention to the distinct cerebellar features in the symptomatology, especially in Dr. Lloyd's cases. It was well known, however, that in Friedreich's ataxia we had a cerebellar symptomatology, in part at least. Dr. Mills thought that Dr. Spiller had not said very much about this, except in referring to Marie's paper.

Dr. S. D. Ingham said that he had recently seen a case that belonged to the same group as those just presented, although it differed from them in certain points, and belonged rather to the type of Marie than that of Friedreich. The patient was a married woman about 30 years old, who, during the past two years, had gradually developed difficulty in walking. The gait was distinctly of the cerebellar type, similar to that of the negro boys shown by Dr. Lloyd. The tendon reflexes were very active, especially in the lower extremities. Ankle clonus and Babinski's sign were demonstrable on both sides. The attitude of the feet in relaxation was equinocavus with dorsal extension of the great toes to a position at right angle to the tarsal bones. Dr. Ingham thought that this case, considered with those presented, well illustrated the variations in the involvement of the various spinal tracts in this disease. In his case there was positive evidence of pyramidal tract degeneration, in addition to that affecting the cerebellar system.

Dr. J. Hendrie Lloyd said, in reference to syphilis, he did not mean to imply that his cases were syphilitic, but he thought that this was a question that should be thoroughly sifted. In reference to syphilis in some members of a family and not in others, he would remind the members of the old law of the attenuation of the virus. That is to say, the first of a syphilitic family would be very syphilitic, perhaps would be stillborn; the next would perhaps break out in syphilis in six weeks. Later children would show syphilitic infection in lesser degree. Jonathan Hutchinson was an advocate of this law of the attenuation of the virus. Dr. Lloyd said he did not believe much in the law, but it only proved that there can be some children more affected than others in the same family. Cases of twins have been reported, born of a syphilitic mother, in which only one of the two children was infected. The tests have not proved that any of the family were syphilitic in the case of the negro boys. In reference to attitude, one boy had the Romberg symptom pretty well developed, the other not. The appearance of slight optic atrophy in both cases is noteworthy.

HEMIPARESIS WITH PARALYSIS OF THE SYMPATHETIC. Presented by Dr. William G. Spiller.

The patient was a boy in whom a hemiparesis had existed from early childhood, and on the side of the hemiparesis the sympathetic supply of the eye was paralyzed. He discussed the representation of the sympathetic brain. The paper will be published in full later.

DISCUSSION

Dr. S. D. Ingham recalled having seen a patient in whom unilateral sympathetic symptoms were apparently due to a lesion above the medulla. A thalamic lesion was indicated in this case by a disturbance of tactile, pain and thermic sensation together with spontaneous pain, all of unilateral distribution. The associated sympathetic symptoms consisted of a small pupil and a narrow palpebral fissure on the affected side, and unilateral sweating of the face and trunk. There was also a slight edema or puffiness of the hand of the affected side. In this case it was apparent that the sympathetic symptoms were not limited to the distribuion of the cervical ganglions since they extended to the arm and trunk. In Dr. Ingham's opinion this case furnished evidence of what Dr. Spiller had emphasized, namely that there is a definite area for the control of sympathetic functions in the region of the midbrain. He thought that it could be placed at least as high as the basal ganglions, probably in the thalamus. The question of the cortical control of the sympathetics is a very difficult one. It is well known that the emotions play an important part in causing sympathetic reactions, but these effects are apparently more dependent on emotional reactions than on the stimulation or the loss of any particular part of the cortex. Experimental work along these lines has not been very satisfactory, as it was difficult to get a fair test by which to associate sympathetic functions with cortical representations.

Dr. S. Leopold said he wanted to emphasize what Dr. Spiller had brought out. In 1916 he presented a case before this Society in which the picture was about the same as that described by Dr. Spiller. It was a case of a young girl with a right-sided hemiplegia in association with jacksonian epilepsy limited to the upper extremity. At that time he noted the features of sympathetic disturbance and looked up the subject and found the same factors that Dr. Spiller mentioned. He had been interested to see whether there was any representation in the cerebrum and exactly what part it had played in this picture, and found that the experimenters had been able to trace the sympathetic pathways only as far as the midbrain. Dr. Spiller asked Dr. Leopold to look up this report of his case and to find out whether it had been reported. He found that it had been reported only by title.

Dr. Charles K. Mills said that in connection with the discussion of Dr. Spiller's paper it was worth while to consider the relations of the basal ganglia, and especially the caudate nucleus, to the autonomic nervous system. In 1914, Dr. Mills contributed a paper on "Emotional Expression, Muscle Tonicity," etc., the main case reported in this paper being one in which Dr. Spiller made the microscopic investigations. The lesion was a markedly destructive one of the lenticular and caudate nuclei, the latter having almost disappeared. There was some evidence of loss of control over the autonomic nervous system in this case.

A CASE OF CEREBELLAR ABSCESS. Presented by Dr. Alfred Gordon.

L. G., a boy, aged 14, came under Dr. Gordon's observation because of an unsteady gait with a tendency to fall toward the right side. Examination revealed a purulent discharge from the right ear of several months' standing. The mastoid process was somewhat tender to touch. A history of otitis media following a protracted course of scarlet fever, preceded by diphtheria, was given. The boy's temperature was then 97.5 F. A blood examination revealed a fairly pronounced leukocytosis, namely, 17,800; hemoglobin, 60 per cent.; red cells, 3,420,000. The spinal fluid removed was bloody, and smears showed pus cells.

The interesting diagnostic symptoms were: Nystagmus was present. It was persistent, its quick movement was directed toward the left side. Standing on the right leg was difficult, but it was easy on the left. There was no Romberg sign. In walking there was a tendency to go toward the right side, and the head and trunk were also slightly inclined to the same side. When the patient, lying on his back, attempted to sit up, he raised his right leg first, while the left remained on the bed. On the other hand, when seated on a chair he was told to raise his right leg, he first flexed the thigh on the pelvis and then only succeeded in elevating the leg; in other words, hemiasynergia was present. In the well-known test for the finger to nose movement, hypermetria was evident in the right hand. It was also observed in the right leg as in each attempt to advance in walking he raised his right foot off the floor higher than normally. Adiadokocinesis, as well as Holmes' and Steward's tests for abnormal voluntary and passive movements, were absent. The knee jerk on the right side was diminished when compared with that of the left side. The toe-phenomenon was absent with all the tests.

The most interesting peculiarity was seen in the past pointing test. With eyes open and facing the writer the patient was told to hold his right index finger in contact with that of the writer. He was then told to close his eyes, lower his arm and again bring the finger in contact to the former position. It was observed that the patient's finger would invariably deviate outward from the examiner's finger. A similar test with the left hand gave a normal result.

The patient's eyes, in addition to the nystagmus, showed a mild degree of papillitis on both sides. The pupils were normal. The ocular muscles were not involved. There was no other cranial nerve involvement. Sensibility of the face was not altered. There was no evidence of paralysis on either side of the body.

The patient complained of frequent vertigo and of constant though mild headache. There was no vomiting during the time of the writer's observation, although the patient formerly had not infrequent attacks of vomiting. Insomnia was persistent. The patient complained of shortness of breath. On examination it was observed that there was a persistent bradycardia. The pulse was 50. The mentality was not impaired.

In making a diagnosis the following possibilities were considered: temporosphenoidal abscess, labyrinthitis and abscess of the cerebellum. The history of otitis media with persistent otorrhea led the aurist to the consideration of a mastoiditis; but the symptoms enumerated above suggested a deep seated abscess in the area of the cerebellum. First, in labyrinthine diseases in which the vestibular apparatus is involved and in which there are also some symptoms common to cerebellar diseases, there are nevertheless present some manifestations which were absent in the latter, namely, Romberg sign; vestibular ataxia, which is, properly speaking, a static ataxia in which individual movements of the limbs are not modified while they are greatly disturbed in cerebellar cases; finally nystagmus usually had a tendency to disappear and it was always directed toward the sound side, while in cerebellar diseases it is constant. (The Bárány caloric test, which is negative in labyrinthitis, could unfortunately not be carried out in this case.)

In cerebral abscess of the temporosphenoidal region nystagmus is exceedingly rare. Equilibrium may be disturbed but it is not of the unilateral character of a cerebellar condition. Asynergia is absent. There is usually some degree of aphasia or paraphasia. There is also deep seated tenderness of the head on the side of the abscess. Mentality is always dull.

In the present case, on account of the special character of the nystagmus, of the hemiasynergy, of the past-pointing, of the dysmetria, strong presumptions were in favor of a cerebellar abscess on the right side, despite the fact that the mastoid process was tender. Accordingly an operation was urged over the cerebellar area, but the aurist insisted on a mastoid operation. A mastoidectomy was performed by him, but no pus was found and no necrotic bone was discovered. He then trephined the temporal region of the right side, but again no pus was found over the temporosphenoid area of the brain. The incision was then closed. The patient's condition became more and more alarming, and he died on the next day. At necropsy a large abscess was found in the right cerebellar hemisphere. The pus contained streptococci (in large chains) and staphylococci.

The case is instructive from the standpoint of the diagnosis as one may be misled by the tenderness of the mastoid bone and consider only a mastoiditis. Necessity is indicated for a careful analysis of symptoms that may be observed in cases of otitis media thus leading to an accurate localization of the disorder either in the labyrinth, in the temporosphenoidal region or in the cerebellum. Exact surgical intervention is dependent on the latter.

ASYNCHRONICITY OF THE DIADOKOCINESIS FROM A MESENCEPHALIC LESION IN THE COURSE OF ENCEPHALITIS LETHARGICA. Presented by Dr. Tom A. Williams.

A woman was seen at Rutherfordton, N. C., with Dr. Henry Norris and Dr. J. C. Twitty, in September, 1919. In March, torpor and diplopia ushered in the attack, the acute phase of which lasted a month. Toward the end of May, hemiplegia occurred accompanied by dysarthria. These conditions had been gradually subsiding, but diplopia persisted except when the gaze was directed laterally. It was due to an insufficiency of convergence and more especially to the weakening of the left internal rectus. When the patient looked downward, however, fusion was possible. The reflexes were increased more particularly in the right lower limb, but both plantar responses were flexor. There was no change in the abdominal cutaneous reflexes. The gait was hesitating and somewhat swaying, and toward the right, increased with the intentional compensation exerted by the patient. The motility was only slightly weakened on the right side. There was no impairment of sensibility even the sense of attitude, and stereognosis being intact. Involvement of the cerebellar system was clearly demonstrated by the disturbances of the patient's balance when the left arm was violently thrown back, and by greater flaccidity in the left arm and wrist and by the greater number of the oscillations of the leg suspended over the side of the bed when the patellar tendon was tapped. The number of oscillations on the right side was five and on the left side seven, before the leg came to rest. These were all common enough differences where there was interference with the efferent cerebellar impulses. They were merely mentioned in order to give a perspective as to the condition present, which made the case worthy of report. The further fact was noted that while on the right side the pronator supinator diadokocinesia was normally rapid, 7.8 per second, the diadokocinesia on the left side, although also of even rhythm, was of greatly reduced rate, about 5.4 per second.

TO ILLUSTRATE THE METHOD OF REMOVING PHOBIA. AGAROPHOBIA COMBINED WITH CLAUSTROPHOBIA. Presented by Dr. Tom A. Williams.

A woman, aged 33, colored, had for eight years been unable to cross a wide street or to remain in a church or theater without intense emotional disturbance manifesting itself as palpitation, polypnea, pallor, chilliness, moisture and cyanosis of the extremities, rigidity and pain in the neck and back, nausea and sensations of weakness and dizziness. Examination showed no physical disease, other than mild hyperthyroidism.

The first occasion on which she had experienced similar sensations had been on a hot summer day, in a poorly ventilated church, when she had felt an overwhelming sense of illness. The compulsion to leave the church had been intense, but because she was seated far up in front and did not wish to create a disturbance she had forced herself to remain. Some weeks later another attack occurred. They gradually occurred more often, but were at first avoidable by sitting near the door. Four years later, because of the sensation of oppressive heaviness which she feared might lead to unconsciousness, she had become afraid to cross a wide space. She had been an exceptionally strong girl, without worries, except anxiety over the health of an invalid mother. She had always feared high places, but thought nothing of it as the other members of her family all felt the same. There was no fear of the dark, no social timidities, other than dislike of being conspicuous as in class recitations.

The diagnosis was that the agarophobia and claustrophobia were hysterical in origin, arising from the powerful suggestion of the recollection of a particular experience, made efficacious now only by the timorous imagination of the patient. Relief of the chronic emotional strain was believed sufficient to cure this. Reeducation was forthwith begun. The patient was unwilling to introspect at first, but yielded when it was explained that to know one's self was as important a part of living as the understanding of technic is necessary to piano playing. It was explained that her dread of a close or open place was due to her ignorance of the mechanism of the consequences of a wrong way of looking at things and the consequent emotions, and that without a true insight into her own psychologic machinery she would be unable to control it. The power of induced ideas was illustrated by the story of the lawyer made sick by a few skilfully implanted ideas, as given in the play, the "Harvest Moon." After a while she accepted this explanation adding that "it must have been fear, for on the first occasion, when leaving the church, one of the maids said: 'What frightened you?'" In the course of a few days she wrote an account of her view of her own psychology in which she showed a clear grasp of the power of conceived ideas, and concluded by saying: "It is difficult for me to understand that these signs of illness are not illness at all but are caused by an induced impression. But now with the assurance that there is absolutely nothing wrong with my physical makeup, my problem seems to be to rid my mind of the fear which has unconsciously but completely controlled my thought."

The next step in the treatment was to demonstrate the truth of this. Dr. Williams accompanied her to a large square and made her cross it alone. Though evidently accomplished under great strain, she declared that she had performed the feat better than at any time since the beginning of the trouble. Later she attended church with only momentary discomfort. Dr. Williams concluded with the final adjuration, that now since she realized she was well all depended on herself.

This violent and persistent long continued agaraphobia and claustrophobia were traced to a single incident on which they were dependent. They were removed in less than a week by efforts directed toward giving the patient an understanding of their mechanism, indeed, compelling her to grasp it and then compelling her to take an exercise which afforded a practical demonstration.

Dr. Alfred Gordon said that in speaking of psychoneuroses, Dr. Williams made the remark that in these cases there was a special makeup of the individual. To Dr. Gordon's mind all psychoneurotics have a special makeup. All of us now and then meet with accidents and frights, and still we do not develop obsessions, but those who do develop obsessions always reveal a special makeup. To say that we can cure phobias or obsessions at one seance, as Dr. Williams had expressed himself, Dr. Gordon could not see for the reasons mentioned how this could be done. Dr. Gordon thought all of the members of this Society had had a great many cases of this character. Some practice suggestion and find that they have the greatest difficulty in removing obsessions and other phenomena of the same nature. Others practice persuasion and still others psychanalysis. In the latter, Dr. Gordon does not always see the sexual element predominating. Dr. Williams practiced analysis of the people in the case he described. He tried to find the psychogenetic element in the case. He made the patient see for herself, he demonstrated to her the cause of her disturbance, and after the patient perceived the fault the trouble was removed. Dr. Gordon calls it persuasion. In regard to the difference in the results from persuasion and suggestion, suggestion was practiced from time immemorial. The speaker believed that suggestion alone in the majority of cases was of no value. In the method Dr. Williams described the patient particpated in the treatment. He laid before the patient the possible causes of the disturbance, and the patient seeing the mechanism, gradually improved.

Dr. W. G. Spiller asked, What was the essential difference between the method as described by Dr. Williams and the method of Dubois in treating the psychoneuroses? There must be psychanalysis in each method.

Dr. W. W. Hawke said that about three years before Dr. Patrick read a most extraordinary paper on this subject under the title "On the Obsessions of Fear," in which he went over this in practically the identical manner Dr. Williams had done. Dr. Hawke said he thought that practically every man had large numbers of these cases. He knew he had. He thought by knowing your case you could select the treatment.

Dr. Tom A. Williams said the method was not in any way especially different from that of Dubois. Dubois tried rational persuasion, but he did not in most of his cases endeavor to reach the groundwork, or mechanism, of the condition and the method was largely one of persuasion. Dr. Williams claimed his method was one in which the personality of the physician had nothing to do with it whatever, except that it was the personality which had taken the trouble to acquire a certain amount of skill in that particular sphere of therapeutics. It was a very difficult thing to do. If it were easy, we should all be doing it. It was done by a great many men in war; some acquired great skill in it. Some men would send back 50 per cent. of neuroses, some 90 and some 100 per cent. and a great many would cure the soldier in one sitting. The phobia is not cured in one sitting, but we give the patient sufficient material so that he can cure himself. It was necessary to go over the laborious processes of so-called psychanalysis in order to cure these patients. Dr. Williams said he had heard Dr. Patrick's paper and discussed it. Dr. Patrick had spoken very well about the fear psychosis, but he did not have success in curing the patient because he failed to cure a great many of them. Dr. Gordon hit the nail on the head when he said that suggestion was a very bad word. As soon as the patient understood the meaning of the fear, he was cured.

DOUBLING OF THE SPINAL CORD WITH PRESENTATION OF SPECIMEN. Presented by Dr. B. Lipshutz.

This paper will be published in full at a later date.

DISCUSSION

Dr. Alfred Gordon said he wished to call attention to a specimen he had exhibited nine years ago before the pathological society. In that case he had diplomyelia and in addition to it he found other conditions, namely, a true porencephaly in the right temporosphenoidal region. He found that the additional cord presented great variations in regard to the size of the cornua, and to the number of cells in each cornu. The original cord presented also malformations in regard to the number of cells, and to the shape of the cornua. The important thing to know was: In Dr. Gordon's case the boy, who was 15 years of age, was perfectly well up to that time and then began to develop epileptic seizures. It is interesting to know that congenital anatomic disorders are more tolerable and produce less disturbance during life than acquired pathologic conditions.

Dr. J. Hendrie Lloyd said that some years ago he had prepared a note on malformations of the spinal cord. It seemed to him that there might be some misunderstanding of terms, but he only looked hastily at Dr. Lipshutz' specimen. It seemed to Dr. Lloyd that this was a bifurcated cord, not a double cord. He thought a double cord was hard to imagine from an embryologic standpoint. Each of these branches is apparently half a cord. Some years ago there were some cases reported of so-called doubling of the spinal cord. He remembered seeing pictures of sections of two cords that were represented alongside of each other. Van Gieson of New York in 1890 published a paper in which he showed that these cases of so-called doubling of the cord were really artefacts, but Dr. Lipshutz' case was entirely different from these, for it seemed to show a branching of bifurcation of a single cord. Of course, the commonest malformation of the cord is that which is seen in spina bifida, and the query arises whether Dr. Lipshutz' case is allied to this malformation. A double monster, in which there is a true union of two distinct cords, must be rare; and still more rare must be a single embryo in which are developed two spinal cords.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, Dec. 18, 1919

George A. Waterman, M.D., *President*

PEARLY TUMORS OF THE BRAIN. Presented by Dr. Percival Bailey.

After some preliminary remarks on the occurrence and pathology of pearly tumors of the brain, Dr. Bailey presented a case in which the tumor, located in the fourth ventricle, was extirpated, followed by complete recovery.

DISCUSSION

Dr. H. C. Solomon: Among our routine necropsy examinations in the A. E. F. we performed one on a young soldier who had been killed by a gunshot wound in the hip which had become septic. On taking off the calvarium we were very much surprised to see a tumor mass which had eroded the skull. There was a definite depression in the brain and the membranes were torn. We thought at first that it was an endothelioma. It was adherent to the skull. When we examined it histologically it appeared to be a cholesteatoma. This was not only my conviction, but it was also the opinion of the other pathologists.

So far as known, there had been no symptoms and as he had been killed by a wound received at the front, it is to be presumed that he was normal in every way.

Dr. Harvey Cushing: I would like to say a word about these pearly tumors. In my brain tumor series there are about 500 tumors that have been verified histologically. The two examples of these pearly tumors described by Dr. Bailey are the first that we have seen. Dr. Bailey has been through the literature very thoroughly and he finds only seven cases. These, however, present exactly the same kind of tumors that he has described. They are very different from the "cholesteatomata" mentioned by Dr. Solomon, which are not particularly rare. There are no cholesterin crystals in these pearly tumors. They are extremely rare, the percentage of incidence being something less than 0.2 per cent. in an ordinary series. Thus the two which have appeared in our series represent about the percentage in which they should occur. As in the tradition of all clinics, these two rare cases have occurred in conjunction.

In general, by far the most encouraging cases are the cerebellar cases, for they may recover completely. The acoustic tumors do not recover so well because there is always a residuum left. Most favorable are the cases of gliomatous cyst. When the cysts are thoroughly evacuated, if well treated, the patient may go on for years. I have now a good many patients with cerebellar cysts, a number have had repeated operations but a considerable number of them in our series are doing well, working and earning their livelihood, ten years after treatment.

A case of cerebellar tumor, I think, has a better outlook than a case of tumor in any other part of the brain. Of course the cerebellum is less vital in many ways, and the diagnosis is usually fairly simple. In the last nine months we have had about twenty-five cases; probably 50 per cent. showed no nystagmus. In the case presented by Dr. Bailey we surmised that the tumor was in the cerebellum, and we found that the patient had an occasional slight nystagmus, but if she had not had other symptoms we would have disregarded it. A great many of these cases have a median cyst, and with a median cyst there is no nystagmus. If the cyst is median, there may be some static instability but incoordination of movement of the upper extremities may be absent.

The most difficult point in diagnosis is to determine whether the tumor is frontal or cerebellar. It has interested me in looking over the report of Horsley's cases in the National Hospital to find in how many cases a frontal operation had been done and postmortem a cerebellar tumor found and vice versa.

BABINSKI'S THEORY OF HYSTERIA.—Presented by Dr. Morton Prince.

The reader began by reciting the fact that of recent years, and particularly as a result of the experiences in this war, there has developed amongst French neurologists, under the teachings of Babinski, a reaction against the classical conception of hysteria of Charcot and his school. The present tendency, or rather the concept which is in vogue, is to regard the classical symptoms such as paralyses, anesthesias, convulsive seizures, etc., as artificially manufactured by the physician or the environment through the influence of suggestion and not as essential manifestations. In this view these maufactured symptoms are identified with hysteria and consequently hysteria becomes nothing more nor less than a group of suggested symptoms. There was evidence that Babinski's teachings were having considerable influence with British neurologists who were engaged in the treatment of war psychoses in the army hospitals. The reader, therefore, undertook an examination of Babinski's views.

Babinski ascribes to hysteria only those "accidents" which have "the common characteristics of being capable of being reproduced experimentally by suggestion" which he avers is "capable of determining the form, the intensity and the duration of them"; and, correspondingly, they can be made to disappear by the influence of persuasion and suggestion. In these accidents or symptoms are included convulsive attacks, paralyses, various contractures, tremors, choreic movements, sometimes irregular but generally rhythmic, troubles of phonation, of respiration, or sensibility (anesthesias, hyperesthesias), and sensorial troubles.

It will be noted that the common mental symptoms, like amnesias, hallucinations, confusions, etc., have no place in this classification. From his point of view, Babinski would abandon the name of hysteria and replace it with the term pithiatism, from the Greek words meaning "I persuade" and "curable," which expresses, in his view, the fundamental character of these "accidents," namely, possibility of curing them through persuasion. Accordingly, he defines hysteria as "a pathological state manifesting itself through troubles which it is possible to reproduce by suggestion in certain subjects with a perfect exactness and which are susceptible of disappearing under the influence of persuasion (contra suggestion) alone."

This conception of a pathologic state, of which the so-called troubles are only manifestations, is a sound one: but after the formulation of this definition we hear nothing more of the pathologic state—only an exposition of its manifestations. This failure to keep in mind, on the part of Babinski, the conception of the pathological state, is the weak point in the edifice which he builds up. Dwelling only on symptoms he fails to grasp the essential problem of hysteria, losing sight of the pathologic state which should be the goal or solution sought. He confines himself to certain physiologic phenomena and loses sight of the fact that many of the so-called troubles which are the manifestations of the "hysterical state" are pure mental stigmas. These are not included in his group of pithiatic symptoms, i. e., are not recognized as hysterical, though they can be induced in favorable subjects by suggestion. Necessarily, therefore, he fails to recognize that many of the cases which he makes use of to support his thesis were already in the "hysterical state" and manifested classical manifestations of hysteria. These cases, he supposes, though having been exposed to emotional trauma, were free from hysteria until they were later the victims of suggestion. The consequence of this lack of vision is that when he comes to his data, to show that hysteria is not induced by emotion but by suggestion, he naively cites cases which every clinician ought to recognize had already developed hysteria and already exhibited classical hysterical symptoms of a mental kind induced by emotional trauma, or mental stress and strain, before suggestion, even in his own opinion, had got in its work.

The reader had no cause to quarrel with Babinski for insisting that hysterical phenomena can be suggested. Every neurologist has seen often enough examples of this, and indeed is aware that he himself has suggested such phenomena, intentionally or unintentionally. Nor can any one doubt that a large proportion of hysterical stigmas, particularly those following traumatisms, have been so suggested, generally unconsciously, by the examining physician. But that all have been so suggested, or what proportion have been so suggested, is another question. Given a certain state of mind, and symptoms can be manufactured almost ad libitum. In such an abnormal state the symptoms can be educated just as by reeducation they can be made to disappear. But what is the state of mind? That is the problem.

You cannot take any man in the street, or even any patient with organic nervous disease, such as tabes, and by suggestion create hysterical stigmas. What is the difference between the normal mental state of the man in the street and that of the person who has undergone some sort of psychologic conflict, or trauma, or shock, that the one is practically immune to hysterical stigmas by suggestion and the other permits them to be created with ease? This is the crux of the problem. As a matter of fact, the hysterical state is a condition of mental dissociation accompanied by certain automatisms due to psychologic factors. The real point, therefore, is whether this state can only occur by the force of a direct suggestion or whether it may be the result of other forces, such as the discharge of an emotion, or by the repressing or inhibited force of other psychologic factors involving a complicated internal mechanism, etc. Babinski rejects emotion as a possible exciting cause and finds that suggestion is the only possible cause left. As one of the links in the chain of his argument he contends that hysterical phenomena never appear at the moment of, or immediately after the emotional shock, when the emotion is at its height, but that always, "between the emotional shock and the presence of hysterical (pithiatic) accidents there is an intervening phase, sometimes quite long, which Charcot called the 'phase of mediation,' during which autosuggestion or heterosuggestion have the opportunity to intervene" and induce the accidents. In support of this view he cites the observations of numerous writers who had the opportunity in this war to observe so-called "shell shock" at the front and behind the lines. Babinski seems at first to be supported by these observations, for it seems that those symptoms of hysteria which Babinski elects to call alone pithiatic or hysterical, that is the paralyses, anesthesias, etc., rarely developed at the time of the emotional shock but appeared after an interval, when the soldier had reached some place behind the lines.

A study of the reports, however, shows that the patients, apparently without exception, exhibited at the emotional period immediately following the shock, hysterical mental symptoms of a very marked character. Among the symptoms commonly described were amnesias, hallucinations, deliria, inability to respond to questions, even when attempts were made to force a response, apparent incapacity to perform a voluntary act, states of hebetude, stupor, confusional states, states allied to fugues, mental dullness, irrational states, tachycardia, tachypnea and tremor.

What are these, it may be asked, with the exception of the three last (which are only the physiologic manifestations of emotion) but states of dissociation with automatisms or unregulated and uncontrolled functioning of disintegrated psychologic systems? They are from the modern point of view typical and pure symptoms of the hysterical pathologic state.

That certain symptoms like paralysis, anesthesia, dumbness, deafness, etc., do not appear until a later period may be a fact, as a matter of observation, but the real question is *why* do these particular symptoms appear only later, while other and mental symptoms appear at the height of the emotional discharge? It is a question of the WHY. The fact which seems to have been brought out by observations during this war is that the hysterical state manifesting itself by mental dissociation, can be induced immediately under mental stress and strain at the moment of the emotional discharge, while certain other symptoms, in the great majority of cases, develop only after a period of incubation.

Babinski's fundamental error is not recognizing that the hysterical state is one of functional dissociation and that any psychologic factor capable of producing such a dissociation, whether it be emotion or a conflict, is capable of producing hysteria. The mechanism by which individual symptoms is produced is another problem. It may be suggestion, as we all know, or it may be a very complicated mechanism which still requires solution.

DISCUSSION

Dr. E. W. Taylor: I presume nearly everyone would agree with Dr. Prince as against Babinski's attitude. It seems to me to be clear, just as Dr Prince has said, that for many years the fundamental idea of psychologic mechanisms have been coming into prominence which lie behind the physiologic symptoms which Babinski has described. In other words, Babinski described, whereas others have done rather more, to interpret.

Dr. Walter B. Swift: I agree with the thesis of Dr. Prince in almost every respect, if by "disassociation" he means an entirely conscious process. I would like, however, to say a word in favor of Dr. Babinski. The background of his original training and his experience has not been chiefly psychologic. As he is principally an internist, his medical attitudes being therefore chiefly observational, he is primarily an observationalist. We would, therefore, naturally expect him to describe observations rather than to create or formulate psychologic concepts of hysteria. He illustrated this attitude clearly when I was in Paris and saw him demonstrate his reflex. He showed three actions besides the extension of the great toe which constituted that reflex. I was more interested in his acuteness of observation than I was in the details of that reflex. Again, how well his attitude of mind is illustrated by his holding, some years ago, that hysteria constituted the symptoms; or, in other words, that the symptoms found in hysteria are the disease.

Further researches are needed today on hysteria. This is a great field still unexplored. Some of you men with a large hysterical clientele in your clinics should take these cases through the steps that I have taken stammering. We medical men first interpreted stammering as being a physical thing; later a vocal diagnosis was made. Finally the diagnosis was traced to the nervous system, until in the end the defect was found to be lodged in the subconscious mentality. At this time I entered the field and discovered the conscious mentality of it. I found a constant conscious psychologic lesion. It was a vitiation of visual processes. The field of hysteria today is ripe for a similar contribution. The conscious mentality of hysteria, decidedly, needs full investigation. With our new modern psychologic tests and recent new medical avenues for conscious mental investigation (found in speech tests) we ought to enter the field of the conscious mentality of hysteria and discover something new, perhaps the real final lesion.

I was interested to hear Dr. Prince present Babinski's interpretation of hysteria as the externalization of will-processes. To me this interpretation is entirely unsatisfactory, if he means by will-processes the conscious choice that has been taught us by James. Zehien once demonstrated in the Charitè Hospital a case of hysteria that simulated tabes, and another case of hysteria that simulated multiple sclerosis. That these two cases could be nothing more than externalization of will-processes is hard even to imagine. I consider hysterical manifestations as the externalizations of a conscious lack—an absence in the conscious mentality, which lets hysterical phenomena appear in a similar fashion as the lack of the central motor neuron lets the pathologic reflexes and spasticity appear. A description of this conscious lack is what we need in hysteria today.

FRACTURES OF THE SPINE. Presented by Dr. W. J. Mixter.

The most important group of fractures of the spine are those with cord involvement, and this paper deals largely with them. Treatment must depend almost entirely on the cord lesion except in so far as the bony lesion has bearing on the cord lesion. Laminectomy with an attempt to remove the cause of injury to the cord or to alleviate the effect of such injury is, of course, the operation usually performed. In view of the pathologic considerations early operation would be indicated in these cases: (1) Partial section of the cord, (2) marked contusion or edema of the cord, (3) pressure on the cord from any cause, (4) bony injury which is likely to cause cord injury in the future, (5) severe cases of hematomyelia. Operation would be contra-indicated in the following cases: (1) Complete section of the cord, (2) moderate angulation without pressure, (3) mild edema of the cord, (4) concussion of the cord, (5) moderate hematomyelia.

Splitting the cord according to the technic proposed by Allen may be performed in certain cases, but without great hope of a successful result.

Early operation is most important and if possible should be performed within twenty-four hours if the symptomatology seems to point to any of the pathologic changes noted above as requiring operation, the most important single indication being increase of symptoms.

The care of the paralyzed patient, whether with or without laminectomy, is difficult and the most careful nursing is essential. The lessons of the war are important and the early establishment of an "automatic" bladder most desirable. The Balkan frame is often of help although as a rule a water bed is sufficient. A plaster jacket is unnecessary in most cases except where the fracture is in the cervical region.

If we are to be at all successful in this most discouraging type of surgery we must make an early complete examination of the acute case. If after careful review of the findings operation seems indicated and the patient is not in severe shock, let the operation be performed at the earliest possible moment.

DISCUSSION

Dr. Harvey Cushing: This is a most difficult subject. I very heartily agree with most of what Dr. Mixter has said. I am glad that he is conservative about these cases. I think most people are who have had a great deal to do with them. The war brought out many interesting things about spinal injuries. The mortality among the cases was very high. The first large group of cases that I saw was during a period of service with the British, particularly after coming in contact with Holmes and Sargent who had been collecting these spinal cases. We must wait until they have had an opportunity to assemble their material and study it before we can have what they have promised to give us, a thorough analysis and study of a series of cases in which every single segment of the spinal cord is involved.

The most interesting cases were the lower cervical transverse lesions. The patients had amazingly low temperatures. Men lay in the wards, conscious, comfortable and with complete anuria for a period ranging from four to five days with a temperature sometimes as low as 90 F. I saw two men, who had a high cervical transverse lesion, with a temperature in the eighties absolutely comfortable and taking nourishment.

Dr. Mixter has spoken of another very interesting group of patients, and to us who had the surgical care of these patients the question which Dr. Mixter has spoken of was brought up, namely, that of keeping them clean. The question of debate was "How shall the bladder be taken care of?" because infection is almost inevitable. I have a friend in the French army who is a genito-urinary specialist and has made a study of the bladder sphincters and their action. His best friend, an aviator, fell and broke his spine in the upper thoracic region. He took care of the patient and would not let any one else touch him. He catheterized him himself with the greatest possible care. The boy died inside of three weeks of an acute surgical kidney. With every possible precaution these infections may occur. A catheter drainage means an infection inevitably. Should we leave these patients absolutely alone and let the bladder distend until it dribbles, a method very strongly advocated by the late Dr. Murphy of Chicago, or should we immediately do a suprapubic operation? One can make a puncture over the pubis with a split trocar so that the tube is introduced with no leaking. The method of leaving the bladder alone and letting it distend was adopted and official regulations were made that such patients should never be catheterized. However, in most instances, some one would be on the ground who would fear to let the bladder distend sufficiently and catheterization would be inaugurated. This particular point has never been satisfactorily settled but the thing behind it of particular interest to me is the fact that the spinal cases, cared for in Dr. Head's special hospital, were patients who had escaped bed sores and cystitis. If they escaped from an infection, these automatic states that Dr. Mixter has spoken of usually began to appear. Some of these patients learned just what stimulus they needed to discharge their own bladder.

Of interest from the pathologic point of view are the observations that Mme. Dejerine has made of the spinal cases which have been under her care at Les Invalides. I saw there a great many roentgen-ray plates of her patients and many of these had begun to show bony deposits in the paralyzed muscle.

I do not know how any more definite rules for operation can be laid down than those Dr. Mixter has given. Each case is a law unto itself. It is difficult to foretell what the injury is. I think we have been misled by the fortunate results of operation in which the recovery has not been attributable to the operation. A laminectomy is quite a difficult procedure. The patients are not in the best possible condition for an operation. If the spinal cord were as accessible as the brain, we should feel much more inclined to operate for contusions. If the brain is treated surgically and the contused area removed by operation, it will recover much more rapidly. If we could get at the cord easily and clean out its area of contusion without, of course, damaging it and could do this quickly, by an operation which was not an operation of magnitude, we would have conditions under which we might wish to operate more often. I find that in the more encouraging cases we are rather apt to refrain from operating for many of them do well. In the more discouraging cases we are also rather apt to refrain from operating for they do badly in either case. This is a general rule to which there are, of course, exceptions.

Dr. William J. Brickley: I think Dr. Mixter is absolutely right in saying hold back until you are sure and operate as soon as you can be sure. These cases as I have seen them change from hour to hour. A case of broken back is plus shock, concussion and great laceration. You wouldn't be justified in operating when the picture is that of a blow across the back, you usually have internal injury as well—for example, kidneys or intestine ruptured. Two or three times I have seen ruptured bladder with broken back. That, perhaps, accounts for some of the urinary difficulty following a case of injured cord. It will be well, therefore, as I see it, not to make a diagnosis on one visit but perhaps in two hours to examine the patient again. As I have seen the necropsies in some of these cases I have been surprised to see how small an injury from the pathologic standpoint it takes to kill a person. In other cases it is surprising to see how much movement we have with a badly damaged cord. In other .words, begin at once to treat your patient. Do not forget that there is always something else besides a broken back, if one can discover a broken back readily with the fingers. Usually the other injuries will have to be treated first. Get rid of the shock, investigate the ribs, the urinary tract and intestinal tract, before you operate for if you do not the best operation will not be of avail. In civil practice we cannot just say operate, as is often possible in a hospital. When you are dealing with general practice people often simply will not permit you to do the thing which offers the best chance for the patient. They have to be convinced. The average man thinks that laminectomy is difficult. Therefore he opposes many such operations. I have been suprised in looking over a good many cases in industrial work to see what good recoveries some apparently complete paralytics have made. In two or three years they are working. We do not operate often enough on this type of case. Dr. Cushing expressed the sentiment when he said certain cases are so difficult that we do not feel like taking the risk. I was interested to look up this matter because I have had many cases of injured backs that I have had to pass on, at one time approximately twenty or thirty cases a day, with the idea of finding out why they could not go back to work, and it was discovered that many patients that were being treated for sprained back or lumbago actually had broken backs ot some degree or other. When a person does not get well and has a persisting train of symptoms, it is just as well to take a roentgenogram.

Dr. Morton Prince: I was intensely interested in hearing Dr. Mixter and also Dr. Cushing and Dr. Brickley because I wanted to learn what the most recent modern experience has been, and what are the conclusions which, in particular, the surgeons of this war have drawn from their experiences. I wanted to see what change had taken place in the point of view. I have been much surprised by the similarity of views expressed here to those that were held in the discussions that took place twenty years or so ago. This whole discussion sounds, in fact, like an echo of similar discussions on fractures of the spine which we used to have then. I, personally, have not had an experience comparable to that of those who have given their views tonight, but in the course of some twenty-five or thirty years' service at the City Hospital, I have seen a good many cases and have taken the responsibility of advising operation in a good many. For a number of years I was intensely interested in the subject. I recall that the question of the advisability of operating has come and gone in waves. Following a time of indifference, there came a great wave of interest accompanied by the hopefulness and fervor for operating. That was just after Horsley brought out his operations on the brain. For a time this wave of hopefulness continued, and then it was followed by discouragement and pessimism. Then another group of younger surgeons came along and this group became enthusiastic, took up the subject again, and operated but with the same results. And so it has continued. I confess, as a result of my experience, I have been left with a feeling of absolute pessimism.

I cannot see from what has been said here that anything has been gained by the experiences of the war to show that if the cord has been injured that anything can be done by operating to restore function. The reader's sole conclusion is that if we are going to operate, we should operate early. Of course, I do not understand that Dr. Cushing adds anything more optimistic or pessimistic.

Concerning the question of diagnosis, as to whether there is a complete or only a partial injury of the cord, we encountered the same difficulty during the years of which I spoke. It is certainly a very difficult matter to tell whether one has a complete or partial injury, and I cannot see that anything has been added to our diagnostic armamentarium. I am glad the point has been emphasized by Dr. Cushing and Dr. Brickley that there are a good many cases of apparently complete and partial injury which afterward have improved, and improved decidedly, without operation having been performed. Therefore it follows that if improvement follows operation it may in no way be due to the latter. It is always a question whether it is not better to give the patient a chance to improve without operating on him.

Dr. Walter B. Swift: It has been mentioned repeatedly that rapid or marked recovery has occurred in lesions of the cord. I would like to ask if there is any new evidence which can more directly than ever before trace these recoveries to the growth of new neurons in the cord, rather than to the recovery of old neurons which have been put out of function?

Dr. A. E. Brownrigg: In a case seen in consultation with Dr. Shea of Nashua six or eight years ago, I made the following observations: The patient had fallen from a building violently buckling his back with consequent fracture. Several laminae were removed. The dura was punctured and beneath the point of injury was a large area of fusiform swelling which proved to be blood clot. It appeared that three fourths of the cord was severed. The operation was finished and the patient was completely paralyzed below the level of the lesion. After several years there was some recurrence of sensation in the lower extremities and also some power of movement. He has since been able to earn something by selling small articles on the street.

Dr. Brownrigg expressed himself as optimistic that the operation had been of decided value in helping the cord to unite.

Dr. E. W. Taylor: As a matter of fact, I think there is no experimental evidence whatever that the cord itself recovers, that the neurons grow through. In those instances in which this has appeared to take place, presumably the regeneration takes place through the cord that remains.

Some photographs were shown illustrating the types of lesion that usually occur. One thing that has been very striking is the fact that there is almost never any subdural hemorrhage. As a matter of fact, traumatism to the spine leads to a central hemorrhage almost always and practically never to a subdural hemorrhage, possibly occasionally to an extradural one. Less conservatism in operation owing to improvement in technic is apparently the watchword of the future.

Dr. W. J. Mixter: I think we may say that in fractures of the spine with or without cord lesions there are very definite surgical indications. Certain lesions definitely call for surgical procedure with a distinct hope of improvement. The great difficulty at the present time is the question of diagnosis of these lesions. Allen's work is positive in regard to the benefit that may be obtained by splitting the cord, but one cannot tell in which case the cord ought to be split. That is one of our main reasons for pessimism at the present time. I agree with Dr. Taylor that a subdural hemorrhage is a rarity. The extradural hemorrhage seems more common, but neither of them are of frequent occurrence.

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A STUDY OF BRAIN REPAIR IN THE RAT BY THE USE OF TRYPAN BLUE

WITH SPECIAL REFERENCE TO THE VITAL STAINING OF THE MACROPHAGES *

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Not a little light has been shed on certain of the phagocytic cells found in areas of aseptic inflammation by the use, in histophysiologic investigations, of the so-called "vital-dyes," and particularly by the employment of the azo dyes of the benzidin series. Tschaschin[1] produced lesions of lymph glands, liver and spleen by burning with a red-hot needle, and introduced trypan blue, and other similar substances, into the circulating fluids of the animals during the period of repair. He showed that the mononuclear phagocytes, which throng the inflamed tissues around these lesions, rapidly became filled with large quantities of the dyestuff, held in the form of numberless minute granules or droplets. Other workers have noted similar phenomena, and it is generally recognized now that these mononuclear phagocytic cells, during a certain period of their existence at least, take up such dyes from the surrounding fluids with avidity, and store them in the cytoplasm. C. C. Macklin[2] has recently studied the inflammatory reaction consequent on injuries of bone in rats which were vitally stained with trypan blue shortly before being killed. He found that, in the tissues immediately surrounding the injured bone (fractured ribs, tibias and femora, and trephined skulls), the dye-containing phagocytes were very abundant and large in stages ranging from the second to the tenth days or later, and he concluded that their mobilization in this region was for the purpose of assisting in the removal of the waste material, the result of the injury.

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[1] Tschaschin, S.: Ueber die "ruhenden Wanderzellen" und ihre Bezeihungen zu den anderen Zellformen des Bindegewebes und zu den Lymphozyten, Fol. Haematol. **17**:317, 1913.

[2] Macklin, C. C.: Bone-Repair in the Rat Vitally Stained with Trypan Blue, Anat. Record **14**:43, 1918. The Development and Function of Macrophages in the Repair of Experimental Bone-Wounds in Rats Vitally Stained with Trypan-Blue, Publication 272, Carnegie Institution of Washington, Contributions to Embryology, No. 27, p. 1, 1919.

These phagocytes of aseptic inflamed tissues are quite comparable to the similar cells which are always found scattered throughout the body under normal conditions and which, though differing morphologically among themselves, yet possess in common a phagocytic potentiality toward "finely particulate matter" which has led to their being grouped under the physiologic term "macrophage" by Evans.[3] The macrophages of inflammation, however, are as a rule more numerous, larger and their phagocytic powers seem to be enhanced.

The vital dye taken up by the macrophages renders them easily visible, and also indicates their phagocytic function, for the view is commonly held that the taking up of the ultramicrons of the colloidal dyestuffs from the surrounding fluids, and the storage of these particles within the cytoplasm in the form of small droplets or granules, is a process analogous to that of the phagocytosis of particulate matter (Evans[3] et al.). The physiologic significance of the reaction of these cells to vital dyes has recently been discussed by Shipley.[4]

Although there is no undisputed theory of the origin of these cells, it seems probable from a review of the numerous articles which have appeared on the subject (in particular some of those of Maximow) that, in areas of aseptic inflammation, such as those mentioned above, the macrophages are recruited from several sources, such as the clasmatocytes of the local tissues, the endothelial cells, certain reticulum cells (such as those of the bone marrow, spleen and lymph glands), the tissue lymphocytes and, in some cases perhaps most abundantly of all, from certain lymphocytoid cells of the blood strained out of the current by the peculiar structural and physical conditions of the inflammatory area.

The work on bone wounds suggested the carrying out of similar experiments on brain tissue, for it has long been known that, in injured brain, large mononuclear phagocytes soon appear and become a conspicuous feature of the repair process; and it appeared more than probable that these cells would show an ability to take up and store the vital dyestuffs. Indeed, MacCurdy and Evans,[5] by injecting trypan-blue into monkeys which were the subjects of experimental poliomyelitis, found that cells (Körnchenzellen, endothelial cells, etc.), staining vitally with these dyes, were present in the brain lesions. They regard the Körnchenzellen as typical macrophages (Evans[3]).

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[3] Evans, H. M.: The Macrophages of Mammals, Am. J. Physiol. **37**:243, 1915.

[4] Shipley, P. G.: The Physiological Significance of the Reaction of Tissue Cells to Vital Benzidine Dyes, Am. J. Physiol. **49**:284, 1919.

[5] MacCurdy, J. T., and Evans, H. M.: Experimentelle Läsionen des Centralnervensystems, untersucht mit Hilfe der vitalen Färbung, Berl. klin. Wchnschr. **49**:1695, 1912.

The brain is a particularly favorable field for the employment of this method, since the tissue is white and contrasts well with the dye; and since it contains no cells (except in the hypophysis and choroid plexus) which under normal conditions takes the dye (Goldmann[6] et al.). Thus any dye which would be taken up in the inflammatory area in the brain would be very conspicuous.

The animal selected was the rat, since it is easily obtained, conveniently handled, and had already been used in the bone experiments. Moreover, the brain is small, and may be removed without trouble. Both albinos and crossed black and white animals were used. They were all healthy, young, and well developed. We obtained them from the excellent colony of the School of Hygiene and Public Health of Johns Hopkins University.

In the principal series (S27) the injury was produced (under ether anesthesia) by plunging an ordinary dissecting needle, heated to redness in the flame of a Bunsen burner, into the depths of the cerebrum, a small hole having first been bored in the skull through the midparietal area, after reflection of the shaved scalp. The needle was withdrawn at once, the tissue not being exposed, as a rule, for more than one-half second. However, there was naturally some variation in the time of exposure. In the first few experiments, too, a slender needle was employed, which produced a slighter injury than that made by the coarser needle used later. Thus the injuries were not absolutely uniform in degree, but this was of little or no importance from the standpoint of the object of the experiments. The hot needle-stab has been used to produce experimental brain injuries by a number of workers, such as Coen,[7] Tschistowitsch[8] and others. Aseptic technic was observed and, with two exceptions (one of which was discarded and the other, S27-41, classed as an abscess) there was no resulting infection. Several animals were killed by the operation, but the survivors showed a very low mortality. A uniform technic of staining was carried out, each animal receiving intraperitoneally 4 or 5 c.c. of a 1 per cent. aqueous solution of Grübler's trypan blue forty-eight hours before being sacrificed, followed by a second dose after twenty-four hours. In those animals which were killed sooner than forty-eight hours after the operation the same procedure was carried out, the dye being given at the proper intervals before the operation. In a few of these cases (shown in the table), at the time of the operation, an additional dose of 1 or 2 c.c. was given. In only two cases did the total dose ever exceed 10 c.c. of dye. Some slight variations from this technic occurred, as shown in the table.

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[6] Goldmann, E. E.: Vitale Färbung und Chemotherapie, Berl. klin. Wchnschr. **49**:1689, 1912.

[7] Coen, E.: Ueber die Heilung von Stichwunden des Gehirnes, Beiträge zur pathol. Anat. u. Physiol. (Ziegler) **2**:107, 1887.

[8] Tschistowitsch, T.: Ueber die Heilung aseptischer traumatischer Gehirnverletzungen, Beiträge zur pathol. Anat. u. zur allgem. Pathol. (Ziegler) **23**: 321, 1898.

The animals were usually killed by rapidly washing out the blood vessels of the brain with warm saline, under chloroform anesthesia, after which a 10 per cent. aqueous solution of liquor formaldehydi was run through for about thirty minutes. In some cases the preliminary washing with saline was omitted and a few cases were fixed by immersion, but those from whose vessels the blood had been washed afforded a much clearer picture of the staining. The brain was then dissected out and hardened in liquor formaldehydi.

The stages examined ranged from the time of injury to the seventy-fourth day, and forty-four animals were used in Series 27. These represented twenty-four stages of repair. Almost all brains were examined in liquor formaldehydi in the gross and with the binocular, both as a whole and split transversely so as to show the longitudinal aspects of the stab. Sketches and full notes were made of the results of these observations at the time, and in every brain used. Most of the brains were then cleared in oil of wintergreen and examined with the binocular microscope. Sections of twenty-four cases, extending through the series, were cut from paraffin blocks, usually so as to show the entire length of the stab, at thicknesses varying from 5 to 15 microns. The thin sections were usually mounted clear, or stained lightly with carmin. Hematoxylin and eosin were often used, and, in special cases, tinctorial reactions were obtained for iron, connective tissue, neuroglia, etc. A few brains, with lesions such as those described, at different stages, were immersed for several days in a fixing solution containing osmic acid, and afterward cut into sections for the study of fat.

For low power work, sections of 25, 50 and even 100 microns were made; these were cleared and mounted without conterstaining, and were used in studying the grosser features of the dye distribution.

A number of brains from additional animals operated on in the same manner were examined in the fresh condition, the tissues of the lesion being carefully teased out in Locke's solution and examined as they were, for the study of the vital staining; or treated with a fat stain, as sudan III or Nile blue sulphate. The teasing method, with the use of fat stains, was resorted to in formaldehyd fixed material as well, and gave some interesting results, as did also frozen sections of such material. An interpretation of any one stage was based on the picture gained from the use of all these methods.

As a vital dye, Niagara blue 3B (No. 46159, National Aniline and Chemical Co.) was used in one case (S27-35, two and one-half hour stage) in two doses of 5 c.c. each, the solution being rather stronger than 1 per cent. The staining was for twenty-four hours. It was more toxic than trypan blue, causing depression. Three other rats, injected with the same solution at the same time, were found dead the next morning.

In a tentative experiment, Niagara blue 2B (same manufacturer) was given in a 4 c.c. dose of 1 per cent. strength. The animal was killed at the end of two days, after twenty-four hours of staining. This dye also was more toxic than trypan blue.

Additional experiments, using nineteen animals, were carried out, in which the operative technic was varied somewhat. In two cases a cold needle was used instead of a hot one. In nine others the hole in the brain, made either by burning or by cutting out with a cold sterile scalpel, was filled with sterile muscle, liver or spleen. Again, in three cases the injury was accomplished by inflicting a sharp blow on the head with the end of a small nail, driven into a wooden handle. This produced either a simple concussion or a depressed fracture, depending on the strength of the blow and the thickness of the skull. In these cases the vital staining procedure was usually the same as in S27; all departures from it are noted. Prolonged staining from the time of operation was tried in two cases, and in two cases (in addition to some of the fresh material) the hot stab operation was done without staining.

The table of material contains a list of these cases. It does not include the fresh material.

Table of Material

Experiment	Stage	Number	Staining		Sections Prepared	Remarks
			Amount, C.c.	Duration, Hours		
I. Hot stabs, vitally stained	0 hours	S27-11	4	24	No	
	0 hours	S27-12	4	24	No	
	1 hour	S27-26	8	48	No	Weakly stained
	1 hour	S27-32	8	48	No	1 c.c. dye, intravenously at operation
	2½ hours	S27-27	8	48	Yes	
	2½ hours	S27-33	8	48	No	2 c.c. dye, intraperitoneally at operation
	2½ hours	S27-35	10	24	Yes	Niagara blue, 3B, 1+ %
	4 hours	S27-28	8	48	Yes	
	4 hours	S27-34	8	48	No	2 c.c. dye, intraperitoneally at operation
	6 hours	S27-24	8	48	Yes	2 c.c. dye, intraperitoneally at operation
	6 hours	S27-25	8	48	No	2 c.c. dye, intraperitoneally at operation
	12 hours	S27-22	8	48	Yes	
	12 hours	S27-23	8	48	No	
	1 day	S27-21	8	48	No	
	1 day	S27-5	8	48	Yes	
	32 hours	S27-44	10	32	No	Died
	2 days	S27-6	8	72	Yes	
	2 days	S27-7	8	48	No	
	2 days	S27-8	8	48	Yes	
	2 days	S27-36	10	48	No	Stabs in both hemispheres
	3 days	S27-37	10	48	Yes	Slight injury
	3 days	S27-39	10	48	Yes	Severe injury
	3 days	S27-49	12	48	Yes	Also used for osmic acid preparations, and spreads of pia and dura

TABLE OF MATERIAL—(*Continued*)

Experiment	Stage	Number	Staining		Sections Prepared	Remarks
			Amount,	Duration,		
I. Hot stabs, vitally stained	4 days	S27-9	8	48	Yes	
	4 days	S27-10	8	48	No	
	5 days	S27-55	10	48	Yes	Transverse sections
	5 days	S27-56	5	24	Yes	Transverse sections
	6 days	S27-13	8	48	Yes	Slight injury
	6 days	S27-14	8	48	No	
	7 days	S27-57	10	48	Yes	Transverse sections
	8 days	S27-29	8	48	No	
	8 days	S27-58	10	48	Yes	Transverse sections
	10 days	S27-15	8	48	Yes	
	10 days	S27-16	8	48	No	
	15 days	S27-17	8	48	No	
	15 days	S27-18	8	48	Yes	
	18 days	S27-43	10	48	Yes	Also used for osmic acid preparations, and spread of dura
	20 days	S27-19	8	48	Yes	
	20 days	S27-20	8	48	No	
	24 days	S27-46	10	48	Yes	Also used for osmic acid preparations, and spreads of pia and dura
	29 days	S27-31	8	48	No	Stab in olfacory lobe
	34 days	S27-2	8	48	Yes	
	35 days	S27-1	8	48	No	
	74 days	S27-4	8	48	Yes	
	24 stages	44 cases			24 sectioned	
II. Cold stabs, vitally stained	2 days	S31-1	10	48	Yes	
	6 days	S31-2	10	48	Yes	
	2 stages	2 cases			2 sectioned	
III. Head blows, vitally stained	18 hours	S28-1	4	18	No	
	4 days	S28-2	4	24	No	Spreads of pia
	11 days	S28-3	8	48	No	
	3 stages	3 cases			No sections	
IV. Muscle insert in hole in brain, vitally stained	2 days	S29-1	10	48	Yes	
	6 days	S29-2	10	48	Yes	
	10 days	S29-3	10	48	Yes	
	3 stages	3 cases			3 sectioned	
V. Muscle insert in hot stab, vitally stained	2 days	S30-1	10	48	Yes	
	6 days	S30-2	10	48	Yes	Spreads of dura
	2 stages	2 cases			2 sectioned	
VI. Liver insert in hole in brain, vitally stained	2 days	S32-1	10	48	Yes	
	6 days	S32-2	10	48	Yes	
	2 stages	2 cases			2 sectioned	
VII. Spleen insert in hole in brain, vitally stained	2 days	S33-1	10	48	Yes	
	2 days	S33-2	3	24	Yes	
	1 stage	2 cases			2 sectioned	
VIII. Hot stabs with prolonged vital staining	11 days	S27-45	10	11 days	Yes	Spreads of pia and dura. Died
	15 days	S27-47	15	15 days	Yes	
	2 stages	2 cases			2 sectioned	
IX. Abscess formation in hot stab, vitally stained	12 days	S27-41	10	48 hours	Yes	Spreads of pia and dura
	1 stage	1 case			1 sectioned	
X. Hot stabs without vital staining	4 days	S27-52	..	..	No	Head injected through aorta with Prussian blue, and cleared for study of vessels
	62 days	S27-3	..	..	Yes	Died while dye was being injected
	2 stages	2 cases			1 sectioned	

GROSS PATHOLOGY

The main line of the description will follow along Series 27, and digressions will be made from time to time to discuss the results of the special experiments. Let us consider first the appearance of a typical, vitally stained brain from Series 27. Such a brain presents a characteristic picture (Fig. 1, twelve-hour stage, and Fig. 2, six-day stage). The injured region is very conspicuous, being intensely blue, in contrast to the normal color of the rest of the brain. In the center the needle-stab appears as a dark blue spot.

On examination with the binocular microscope the center of this blue area, usually from 1 to 3 mm. across, is seen to be irregular, somewhat depressed, and typically of a dark grayish blue. It represents the seared and coagulated brain substance; we have termed it the "coagulum." Almost always the upper part adheres to the dura, and on being separated, an irregular surface is left. The coagulum has shrunk away from its surroundings during the process of its formation, and thus we find it enclosed by a narrow, lightly-staining belt, which we have termed the "intermediate zone." Outside of this, again, is a ring, forming the wall of the lesion, which stains intensely blue. This, too, is composed of tissue killed or damaged by the heat. It presents a ragged surface, where the coagulum has pulled away. To it we have applied the name "necrotic wall." These details are not shown in Figures 1 and 2. Beyond the necrotic wall, and rather sharply delimited from it, is a "peripheral zone" of pale blue (well seen in Fig. 1), which gradually fades out into the surrounding normal brain tissue. It varies somewhat in extent in the different brains, at the same stage, and is present only in early stages.

Hence the upper end of the stab presents a series of concentric, approximately circular zones, differing in intensity of staining. The regions termed the coagulum, the intermediate zone and the necrotic wall correspond respectively to what Coen[7] has described as the zones of "necrosis," "fatty degeneration" and "inflammation."

Often numerous dark blue spots are seen with the binocular on the surface in the vicinity of the stab. These are pia-arachnoid cells which have taken the vital stain. It is evident, too, that the superficial blood vessels on the injured side, supplying or draining the traumatized area, stand out by reason of their marked blue color in sharp contrast to the corresponding ones on the opposite side, in which the stain is often scarcely perceptible. Normally, as is well known, blood vessels stain quite plainly and diffusely with trypan blue, but here the staining is much increased over normal. The dura, too, in the vicinity of the stab, shows a strong blue staining, and frequently vitally stained cells may be observed in it.

If such a stab is cut transversely into slices the deeper portions show the same features: a central dense blue coagulum, often containing blood clot in the needle track, surrounded by a blue necrotic wall, sharply delimited from the more peripheral pale blue zone. As on the surface, we find interposed between the coagulum and the necrotic wall a loose fragmented zone, frequently containing blood clot.

These transverse slabs, when cleared in oil of wintergreen, show the different zones very distinctly (Fig. 4, from the six-day stage). The staining in the central plug of coagulated tissue is found to be fairly homogeneous, but blue granules may be made out in it with the binocular during the first day or two, which represent diffusely stained dead cells. A little later vitally stained macrophages are found. Similar blue-stained cells in the necrotic wall are found in stages from the first to the sixth day or later. The masses of extravasated blood are rendered very conspicuous. Often one can see that the blue cells follow the lines of the blood vessels. These are vitally stained cells of the endothelium and of the adventitia; it may be, too, that they are ameboid phagocytes which have wandered to these vessels. They traverse regions pervaded by a diffuse blue coloration; in Figure 4 they may be seen to radiate from the stab region. In pieces from the cortex they give the effect of a forest of slender straight trees as they follow their direct courses perpendicular to the surface.

Thus a well defined color pattern is presented by the upper end of the stab, and by cross sections of it, the elements of the picture corresponding to the different parts of the lesion.

By splitting the lesion longitudinally, the above mentioned regions are revealed in their full extent (Fig. 3, twelve-hour stage). Such sections were usually made transversely to the long axis of the brain. The vitally stained traumatized area is even more conspicuous than when seen from the top, and the contrast with the rest of the brain is very marked. The entire injured region, bluish in staining, is in shape like a truncated cone (as Coen has described it), with its base at the brain surface and its apex corresponding with the region around the point of the needle at its position of maximum penetration. Occupying the center, appears the long narrow wedge of coagulum—ragged and dirty blue—tapering from the upper surface of the brain toward the base, through the cortex, corpus callosum, and often thalamus, lateral ventricle and choroid plexus. The interior of the coagulum, marking the path of the needle, contains extravasated blood, and this has oozed into many of the spaces of the coagulum and the surrounding loose intermediate zone. Outside of the latter region appears the densely blue necrotic wall of the stab, the upper end of which was seen on the surface as a blue ring. As on the surface, it is sharply delimited from the tissue around it by reason of its more intense staining. Medially it presents a ragged surface. As before the binocular microscope discloses in it blue dots, which prove, on examination with the compound microscope, to be dead nerve cells, as well as vitally stained living macrophages. Peripherally there is a variable amount of diffuse pale staining, widest at the top, which, as on the surface, fades into the surroundings.

By clearing a thick slice from such a preparation the dye deposits are vividly brought out, as in Figure 5 (six-day stage). Figure 6 presents a small area from this field, greatly enlarged. Here we see many dead cells and some living, vitally stained macrophages. In the sections of this period there are not found nearly so many stained macrophages as such a slice of cleared tissue would lead us to expect. In the lesion itself there are hardly any. There is much diffuse blue, due to the collection of dye in the exudate. Lines of blue cells along the blood vessels are seen, like those in the cross sections.

These findings are best understood by an analysis of the factors concerned in the production of the injury. During the process of stabbing, the upper end of the lesion was subjected to greater heat than the lower end, for not only did a wider part of the conical needle come into contact with this area, but it was exposed to the entire length of the inserted portion of the needle, both while entering and leaving. The hottest part of the needle was applied here also, for as it penetrated it became progressively cooler. Hence it is evident that the upper portion was more severely burned than that lower down, so that the conical shape of the injured area is explained.

The effect of the heating is to sear a cylinder—or rather a cone—of tissue, immediately about the needle. This "coagulum" pulls away from the surrounding parts, into which steam is forced. Thus a looser area, which we have called the intermediate zone, is produced, and into it fluid exudate and blood penetrate. Around this intermediate zone the brain tissue is severely injured by the heat, and has been called the necrotic wall. Its vessels quickly become thrombosed, as was demonstrated in a cleared specimen the vessels of which were injected with Prussian blue. The central mass is thus temporarily deprived of blood supply. In the necrotic wall the damage, of course, is greatest next to the center, and outwardly diminishes gradually.

These injured parts soon become flooded with tissue fluid laden with the dye, and rapidly absorb it. Many authors (Goldmann,[6] MacCurdy and Evans[5] et al.) have shown that dead tissue takes up the dye and stains diffusely; indeed such behavior on the part of cells toward the dye has been looked on as an evidence of death. Thus it was to be expected that the injured area would be quite intensely blue, and when the relative densities of the different zones to which we have referred are considered it is quite in order that most of the staining should be found in the necrotic wall, and in the coagulum. The material stained appears not to be fat, but the actual protoplasm of the damaged cells.

In addition to this diffuse staining of the dead tissue there is to be considered, during a limited period, as we shall see, a slight staining of the macrophages, most marked about the second and third days, which intensifies very slightly the coloration.

Staining Variations with Stage of Repair.—Having gained a definite conception of the macroscopic characters of a stained injured brain we are in a position to compare the different stages, in order to study the variations in coloration at the successive repair periods. A careful inspection of the upper ends of the lesions and split sections in the entire Series 27 shows that the staining of the injured tissue very rapidly reaches its maximum brilliancy. Fairly bright at the one-hour stage, and quite well marked at two and one-half hours, it appears at the sixth hour almost as strong as at any later time. All the zones are very clear cut at this stage. The stain remains practically unimpaired until the end of the third day, following which it gradually fades, and by the tenth day is quite weak. After this stage it is practically negligible, although faint traces were noted as late as the thirty-fifth day; several earlier specimens, however, did not show any staining, and none of the later ones did. The period during which the necrotized tissue stains with trypan blue, then, extends from the first hour, or even before, to about the tenth day.

This survey reveals a very rapid diffusion of the dye into the necrotic tissue, for in six hours the staining has practically reached its maximum, as far as can be ascertained from inspection. The subsequent lessening of staining intensity may be laid to the gradual absorption of stainable material. In support of this we note that the sections show a progressive rarefaction of the injured tissue. Perhaps a less important factor in this diminution in staining power is to be found in the changes occurring in the dead tissue, which prevent its taking up the dye. The finding of unabsorbed remnants of injured tissue as late as the sixty-second day — long after any diffuse blue staining is visible—favors the latter view. It is not improbable that the composition of the necrotized tissue is gradually changed, through loss of the more soluble albuminous materials, leaving the more stable elements, which do not take up the dye readily. On the whole, however, the use of the vital dye affords a graphic and reliable picture of the process of resorption of the moribund tissue.

If the concentration of dye is increased, the staining, as would be expected, is more dense. Thus in a special case at the two and one-half hour stage, where a solution of dye was used in strength greater than

1 per cent., the staining was more dense than in the other members of the same stage. The amount of stained tissue, of course, varies directly with the severity of the injury. However, even where the operative and staining technic was uniform, differences in the intensity of staining of the lesions in different animals were sometimes noted, these being directly related to the general staining of the animal. When the body tissues as a whole are lightly stained the injured parts are also. Allowance was made for this in making generalizations.

It is interesting to study this staining as it occurs in the different zones of the lesion. In the coagulum the color was quite well marked in the one-hour stage; with further exposure to the dye the color became rapidly intensified up to the twelfth hour, remaining uniformly bright until the third day, and then diminishing progressively until the tenth day. Though traces were present up to the twenty-fourth day, they were exceedingly slight; indeed the coagulum can scarcely be said to be stained after the tenth day. Thus the staining of the coagulum, in respect to its onset, duration and disappearance, follows the scheme of the staining of the entire lesion, and the same interpretation may be accorded it.

The staining of the necrotic wall is quite comparable to that of the coagulum; it is a little more marked in the early stages—probably because of easier access to the dye through intact blood vessels. Then, too, the vital staining of the macrophages on the second and third day, which will be referred to later, is a source of some slight accession of color in the necrotic wall at this time. At many later stages, such as that of the third day, after the dye has had time to diffuse into it, the coagulum has stained more intensely than the necrotic wall. There is probably then less stainable material in the necrotic wall, for here, no doubt, absorption commences sooner, due to better vascular organization. In the necrotic wall, too, the granules due to dead nerve cells are usually more distinct, as cleared specimens show, than in the coagulum, since in the latter the individual cells are, as a rule, destroyed beyond recognition.

The staining in the "peripheral area" is much paler than that in the coagulum and necrotic wall; indeed the contrast is quite striking (Figs. 1, 2 and 3). Here there are fewer dead cells, although these are fairly numerous, especially in the border zone between this region and the necrotic wall. This border zone, as will appear later, marks the limit between the tissue which will remain and that which will be absorbed. Sometimes this pale staining involves the opposite cerebral hemisphere, an area adjoining the stained region of the injured side appearing blue, as in the two and one-half-hour (S27-35), six-hour (S27-24), and two-day (S27-8) stages.

This staining develops almost coincidently with the staining of the necrotized tissue; but very little of it exists after the fourth day, and almost all, if not all, traces have disappeared by the tenth day. A graphic illustration of its disappearance is afforded by comparing Figure 1, of the twelfth hour, with Figure 2, of the sixth day.

It is difficult to explain this peculiar coloration. We may assume that it is due to some change in the tissue, which temporarily lowers its vitality, permitting the cells to take up a little dye; and that they subsequently recover from this injury, with the exception of the cells actually killed in the vicinity of the lesion. This change may be supposed to be in the nature of a shock, and a temporary disturbance of the equilibrium of the protoplasm of the cells, which disappears later. If this were true one would look for dye within the cells proper, and consequently a granular picture would present itself in the cleared preparation under the binocular microscope. This we do not find, except in dead cells in the region adjoining the necrotic wall. Then, too, the sections reveal in this zone only a very little intracellular dye. This is found in a few neuroglia cells, and is quite inadequate to account for the color.

It seems to us, therefore, that the most plausible explanation for this pale staining is that it is due to an edema of the tissue about the injury, consequent on the inflammatory reaction, and that the intercellular spaces are charged with the dye, rather than the cells. Fluid undoubtedly exudes from the blood vessels of such a region, and since this fluid is loaded with the dye, the latter becomes sufficiently concentrated to be easily visible.

It is noteworthy that the tissue in which the stain appears does not become completely absorbed, as the necrotized tissue does, but retains much of its character as nerve tissue, although in the neighborhood of the lesion many of the cells are killed and shrink, losing their normal appearance. Then, too, the tissue around the necrotic wall becomes more porous and open in texture than that of the normal brain tissue, and it is certain that it undergoes much change, as will be seen on reference to the sections. The presence in this area of trypanophilic neuroglia cells points to the presence of fluid exudate.

A similar pale staining was found in the brains of animals that had received a blow on the head (S28) thus, producing a concussion of the brain, or even a lesion due to a depressed fracture. In S28-1 (eighteen-hour stage, one injection of 4 c.c. dye for eighteen hours) the cortex underlying the area of impact showed a distinct, though very pale, diffuse staining. In S28-2 (four-day stage, one injection of 4 c.c. dye for twenty-four hours) the surface of almost the entire cerebral hemisphere was of a pale diffuse blue, more marked than in the eighteen-hour stage. It is associated with a staining of the pia-arachnoid cells and the blood vessels. There was a distinct lesion of the brain in this case, due to the penetration into the cortex of a spicule of bone from a depressed fracture, and here we find the same dense staining of necrotized tissue that was noted in the coagulum and necrotic wall of S27. There were some slight hemorrhages over the cortex. In S28-3 (eleven-day stage, 8 c.c. of dye in two injections, forty-eight and twenty-four hours before killing) there was no staining.

In these cases it is probable that the mechanical effect of the shock caused an inflammatory edema, as in the hot-stab cases, and that the staining arose in the same way.

A similar pale diffuse staining was also noted in the brain and cord in rats that were the subjects of experimental meningitis. The cause was doubtless the toxins arising from the infection, for the staining was obvious for a considerable depth under the infected meninges.

We may refer briefly to the staining of the blood vessels surrounding the lesion, in S27 (seen on the top of the brains in Figures 1 and 2) as revealed by a comparison of the different stages of the series. This staining comes on rapidly; at the one-hour stage the vessels are quite markedly blue, and at two and one-half hours the coloration is as bright as at any subsequent stage. It remains brilliant until the fourth or fifth day, rapidly diminishes, and is not found after the tenth day. It involves the efferent and afferent vessels. In cases where the diffuse pale blue extends over to the opposite hemisphere (as in S27-24) the vessels of this side appear stained also, but not so brightly as on the injured side. The middle cerebral artery, being large and superficial, is usually the most brilliantly stained vessel. This vessel staining was noted in one depressed fracture case (S28-2), of the fourth day.

The staining of the vessels is thus synchronous with the general staining of the lesion, and it seems to be intimately linked with this, and with the inflammatory process. It is certain that in the vessels of the inflammatory region some change has taken place. This change may involve the permeability of the wall, permitting the passage of exudate into the area of inflammation or promoting absorption of the soluble exudate. Histologic examination disclosed nothing beyond a deepening of the usual diffuse blue staining of the wall.

We may here say a word as to the staining of the pia-arachnoid cells, as they are seen on the upper surface of the brain, in the neighborhood of the lesion, under the binocular microscope. They were not noted until the sixth hour. This may be because not enough dye was present in the cell before this to be visible, or that there is a latent period between the application of the stimulus and the onset of dye ingestion. It is noteworthy that the pia-arachnoid cells of the brain of the rat do not, under normal circumstances, stain. Stained pia-arachnoid cells in the neighborhood of the lesion were found in practically all cases from the sixth hour to the sixth day, and they were noted in many, though not in all, cases later than this, up to the twenty-fourth day. Occasionally they were quite remote from the lesions. In a depressed fracture case of the fourth day (S28-2) we found similar stained pia-arachnoid cells in the traumatized area.

These cells, like the phagocytes within the brain substances, are evidently part of the organization for getting rid of waste material in and around the injured area. They will be considered further in the study of the sections.

In stages ranging from the first hour to the sixth day the choroid plexus on the injured side was stained more intensely than that on the normal side. This is not surprising, in view of its proximity to the injury; frequently it shares in this. For the same reason, and also because of the stained macrophages it contains, the dura around the wound is darker blue than elsewhere.

The binocular microscope reveals a very interesting condition in the peripheral zone of many of the cleared slabs, which has already been referred to. Striking rows of cells are seen, which were found to trace out the course of vessels (Figures 4 and 6). These were very conspicuous in the cortex, as well as in the deeper brain substance, and were noted at stages of the first (S27-5), second (S27-7, S27-8), third (S27-37, S27-39), fourth (S27-9) and sixth (S27-14) days. Not only phagocytic endothelium, but perivascular cells as well, are thus brought out. More will be said as to this interesting condition in the description of the sections.

HISTOPATHOLOGY

In the study of the sections an account of a typical lesion will first be given, followed by a brief survey of the principal changes occurring as a result of the processes of inflammation and repair. The macrophages, which appeared in injuries of all kinds, as we expected, will then be described, especially with regard to their vital staining features.

Typical Lesion.—If from an animal of Series 27, killed shortly after the operation, before the inflammatory reaction has become evident, a brain section be so cut as to split the stab, the picture presented will recall that seen in the gross preparations. Figure 12, a low power from the six-hour stage, presents a typical appearance. In the lesion the various concentric zones already described are seen. The coagulum is somewhat homogeneous and glassy in appearance, and often more or less broken. In it are the remains of seared cells, fibers and extravasated blood. In places the microscope discloses areas which are finely reticular in structure. The dead cells and fragments thereof stain a dense blue.

In the intermediate zone the loose, open spaces contain serous exudate and blood, but there are relatively few cells in the early stages. In the necrotic wall, dead nerve and neuroglia cells are found, stained diffusely blue, and showing little or no cellular detail. The inner margin of this wall is irregular and, of course, presents the greatest amount of cell damage. Here the blood vessels are thrombosed and extravasated blood is abundant. Farther out the injury becomes progressively less, leading into the narrow transitional zone bordering the peripheral area. Here it is seen that the ganglion cells, being more delicate, are killed farther out in the nerve tissue than the neuroglia and vascular elements. This feature was very marked in the cortex and in the layers of the cornu ammonis. These dead cells dwindle away as we range out into the peripheral zone, which passes insensibly into the surrounding normal nerve tissue. These features, of course, vary somewhat with the severity of the injury. Thus when a small needle is used, or one relatively cool, the damage is less and the lesion smaller.

Survey of the Healing Process.—As we glance over the sections representing the successive stages of healing it is not long before we are able to recognize marked changes which are well demonstrated in the photomicrographs, Figures 12 to 21, from Series 27. With advance of inflammation and repair the early morphologic features of the lesion are gradually lost. The injured area rapidly becomes invaded by granulation tissue, coming from the surrounding blood vessels and from the pia-arachnoid. Young vascular sprouts, fibroblasts and a few polymorphonuclears soon appear, and a multitude of mononuclear phagocytes quickly takes possession of the entire area. The coagulum and necrotic wall, with the absorption of their substance, melt away, becoming more open, their spaces affording growth channels for fibroblasts, young vessels and phagocytes. The characteristic zoned pattern is thus speedily obliterated.

Soon a sheet of cells forms between the necrotized elements, destined for absorption, and the surrounding damaged, but not completely destroyed brain wall, which is to be conserved. It is represented at first by an aggregation of fibroblasts and other elements, among which are many mononuclear phagocytes. From this mass a definitive membrane is gradually condensed. By the sixth day its outlines are becoming visible, and it was well marked at the fifteen-day stage. It is directly continuous above with the pia-arachnoid (Figs. 15, 16, 18, etc.), and gives the impression of having grown down from this membrane. Fibroblasts from the surrounding blood vessels doubtless contribute to it, and it may be that the neuroglia reinforce it by building up a sclerotic zone around it, as Tschistowitsch[8] believes. Coen[7] derives it from pia, perivascular and neuroglia elements. This membrane, which is sometimes thin and poorly marked, is simply the peripheral border of the core of loose fibrous tissue which comes to occupy the brain defect, and which eventually replaces most, if not all, of the necrotized tissue. The spaces in the scar, which are numerous, and apparently intercommunicating, seem to be continuous outwardly with the subarachnoid space, and are thus probably filled with cerebrospinal fluid. The blood vessels in it, which at first are abundant, affording a thoroughfare for waste product removal, gradually disappear. The mononuclear phagocytes, so numerous in the early stages, slowly decrease in number, but were found in the latest stage examined — that of seventy-four days.

The brain tissue outside of this membrane shows evidence of damage which varies inversely as the distance from the needle. In early stages the inflammatory process shows the usual vascular and cellular changes. Neuroglia and endothelial cells are awakened into phagocytic activity which, as will be seen, shows itself in the appearance of dye granules in these cells in the vitally stained animals. This is the region which has been referred to as manifesting a pale diffuse staining with trypan blue. After the inflammatory conflagration has swept through this zone there persists a residue of the original tissue in which the damage is greater or less according to the distance from the stab. We have already noted the presence of dead cells, especially near the needle track. These cells shrink, show degenerative changes histologically and, in the early stages, stain densely with trypan blue, including the nucleus. Their attenuated corpses may remain in situ for weeks and even months. The intercellular substance shows a porous and reticular condition (Figs. 24, 26) ; the meshes of this reticulum are largest next to the separating membrane, and gradually become finer peripherally, passing over into the normal nerve substance.

Some of these photomicrographs require special elucidation. Figure 14 reveals the characteristic appearance of the three-day stage, the coagulum and extravasated blood being plainly shown, with the conspicuous agglomeration of macrophages between the coagulum and the edges of the necrotic wall. Figure 15 (fifteen-day stage) shows a case where the injury was relatively slight, and healing correspondingly rapid. Hence most of the dead tissue has been absorbed, the defect being occupied by large open spaces, with a reticulum of scar tissue containing a little débris and some macrophages, the whole bounded by a well marked membrane. In Figure 16 (twenty-four-day stage) healing has not progressed so far, probably because the injury is more extensive than in the preceding stage, so that the edges of the wound are not so definitely outlined, less absorption has occurred, and the membrane is not so well developed, although it can be seen at the upper part of the photograph lining the right side of the coagulum. Macrophages and fibroblasts which have grown into the coagulum at this stage of healing are pictured in Figure 17. When we turn to Figure 18 of the sixty-two-day stage we find that some shreds of tissue still remain, pointing to the fact that not all of the defunct brain substance is speedily absorbed. This is in harmony with the findings of Coen and others. The defect, however, is principally filled by a mass of very open reticulum, in which are caught blood cells and macrophages. The shrinking of this scar tissue, with a gradual inpushing of the surrounding brain walls, doubtless reduces the size of the lesion, but in no stage examined was the defect completely obliterated, and as late as the sixty-second day it was quite large. In the more severe injuries the defect probably is never filled. The membrane of pia-arachnoid cells is well formed, and has completely isolated the débris to be absorbed from the surrounding brain tissue. It is shown under high power in Figure 19. The end stage of the process is seen in Figure 20 of the seventy-four-day stage, where the membranes of the two sides of the wound are practically in apposition. This case, however, is from a slight injury, the wound having been made with a small needle.

A few stabs (fifth, seventh and eighth day stages) were cut transversely. These show the various zones most strikingly. The coagulum may be seen gradually breaking up, the fragments becoming enmeshed by the ingrowing granulation tissue. The lesion is filled, in early stages, with a dense plug of cells, which is quite sharply differentiated from the surrounding brain wall. The inner border of the peripheral zone appears as a distinct narrow strip of coarse reticulum, in which cells are very scarce.

Vitally Stained Granules in Coagulum.—The diffuse staining of the dead tissue has been described with the gross material, and it is much more satisfactorily studied there than in the sections. It is possible to make out a distinct staining of the coagulum and necrotic wall, even in thin sections, in the early stages, but in no case was the faint staining of the peripheral zone appreciable in the sections. There is, however, an interesting feature of the staining of the coagulum which should be described here. It consists in a multitude of small bright blue granules, in appearance quite like the "dye granules" found within the cytoplasm of macrophages, which occur among the débris of nerve cells, fibers and other material composing the coagulum (Fig. 27). These granules are usually small, round and scattered singly or in small clumps. They vary in size from those so small as to be seen with difficulty with the oil immersion to aggregations of dye measuring about one fifth of the diameter of a red blood cell. Such large granules, however, are rare, most of them being very small. They are intensely stained, and stand out in sharp contrast to the rest of the coagulum, which shows the usual diffuse coloring of dead tissue. They do not occur in all parts of the coagulum, but only in certain loose areas, reticular in structure, and the appearance is as though the granules were caught in the meshes of this. They are not found in the necrotic wall nor in the outlying brain substance. They were first seen at the six-hour stage. Earlier than this the dye has apparently not had time to concentrate sufficiently to render them visible. In the one-day stage they are more numerous, and in the two-day stage the number is still greater. Traces were noted at the third day.

Thus we have occurring in dead tissue a phenomenon — that of the segregation of the molecules of the dyestuff, trypan blue, to form distinct granular masses, easily seen with the higher powers of the microscope — which we have heretofore been accustomed to associate with the activity of living protoplasm. These granules are probably minute stained masses of protoplasm, highly concentrated, due to the action of the heat, and of such character that they absorb the dye with great avidity, and are thus able to accumulate so much of it, by apparently purely physical means, that they appear very distinct.

The fact that they become progressively more and more numerous from the sixth hour to the second day suggests that the protoplasmic granules, which appear to form the basis of them, vary in their ability to attract the dye, so that the more avid granules are stained first. They occur in the special cases (later to be described) where the dye was given at the time of operation and the animal allowed to live on until the eleventh or fifteenth day; here absorption of the coagulum has evidently been delayed. The fact that they do not occur in Series 27 after the third day suggests that the basis of their formation, the granule of concentrated protoplasm, is speedily absorbed in the normal repair process, or is so changed that it no longer takes up the dye.

In the two-day stage we have the opportunity of comparing these granules with the intracellular granules of the living macrophages which have now invaded the coagulum, and the resemblance is striking indeed; it is quite probable that this would not be so close in fresh material. The granules are never scattered over open spaces in the coagulum, as would be the case if they were simply minute coagula precipitated from the plasma, but always appear in the solid, though tenuous, remains of the coagulum.

In their genesis they seem to be extreme cases of the same process which appears in the absorption of dye by any dead or inert protoplasm; they are thus distinct in this respect from the granule which occurs within the cytoplasm of the macrophage, which (according to current hypotheses) is formed by a phagocytosis of the dye and a concentration of the same within definite areas of the cytoplasm. It is of interest to point out, however, that we have here a distinct type of dye granule formation which cannot be attributed to cellular activity. Up to this time dye granules have been intracellular, whether formed by the staining of preformed material, or by the so-called "vital activity" of the cell expressing itself in phagocytosis. These observations, although brief, serve to record the extracellular occurrence of trypan blue in granular form.

THE MACROPHAGES OF BRAIN WOUNDS

General Survey.—We come now to a consideration of the mononuclear phagocytes which play such an important rôle in the process of brain healing just described. A complete histologic examination of these cells is not intended, and is quite unnecessary, since the literature contains so much regarding them (e. g., the work of Alzheimer[9] and, more recently, of Essick[10]). In this article, therefore, we shall content ourselves with but a cursory notice of some of the more salient features of the cells, passing on rapidly to a discussion of their behavior toward trypan blue, in the experiments carried out. Their general characteristics were found to correspond to the abundant descriptions given by the many authors who have written on the subject, and who have referred to the cells by the term "Körnchenzellen," and by various other designations.

They were not conspicuous in the brain tissue until the second day after the injury, though it is probable that their precursors were on the field some time before their phagocytic activities were so pronounced as to lead to their detection. They increased very quickly, and by the third or fourth day their numbers had reached the maximum. Following this they diminished gradually until the last stage examined, that of seventy-four days, when they were very few. It is possible that they remain in the defect even longer than this. There is some variation in this general rule, as, for instance, fewer were found on the fifteenth day than on the twenty-fourth, but this is due, doubtless, as we have already mentioned, to the relative lightness of the injury in the former case.

As to their position in the lesion, at the time when the first cells were found, on the second day, they inhabited the loose zone around the necrotic plug, and also the necrotic wall. It would seem that they are ameboid, and have wandered into these regions from the surrounding tissue. They are to be found in the neighboring brain substance also at this stage, scattered among the cells and fibers (Figs. 13 and 24). As the coagulum softened they gradually invaded it, as seen on the tenth day; indeed quite a number were found in it on the third day. Sometimes the macrophages occurred in closely packed masses — the "Körnchenkugeln" of Gluge[11] — giving the impression of a syncytium. As the limiting membrane which we have described forms, most of the phagocytes are found in the space which it encloses, and after it has become well marked there are few or no phagocytes to be found in the outlying brain tissue. Often the phagocytes are in the membrane itself. With the disappearance of the débris the macrophages decrease in number and are found in the meshes of the scar tissue, occupying the defect.

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[9] Alzheimer, A.: Beiträge zur Kenntnis der pathologischen Neurologia und ihrer Beziehungen zu den Abbauvorgängen im Nervengewebe, Histol. u. Histopathol. Arb. über die Grosshirnrinde (Nissl), Jena **3**:401, 1909.

[10] Essick, C. R.: Pathology of Experimental Traumatic Abscess of the Brain, Arch. Neurol. & Psychiat. **1**:673, 1919.

The relation of the macrophages to the blood vessels traversing the blue-clouded zone of inflammation is worthy of note, and has been referred to. Hypertrophied endothelial cells, so well known to pathologists, were often seen in the early stages, as at the third day. Frequently macrophages were found in the adventia and perivascular spaces. Often they occurred closely applied to the outside of the vessel wall, and even extended along it. This relationship, in the vitally stained specimen, led to the vessels being outlined by blue cells, so strikingly seen in the cleared slabs. When the capillaries invade the coagulum these cells can be seen composing their walls or closely applied to them, as well as lying free in the perivascular spaces and beyond. This important relation will be discussed at greater length with the fresh tissue.

The average size of the macrophages at the different stages of brain repair was determined by selecting ten representative cells in a section from each of the fifteen most representative specimens, extending throughout S27, and making careful measurements of the longest diameter by the aid of the camera lucida; an average was then taken. This method was fairly accurate, since the shape of the cells examined was approximately the same. No very wide variation in size due to the stage of repair was noted. When the cells are proliferating rapidly, as at the third day, the average size is somewhat smaller (11.42 microns). As mitosis becomes less frequent in the succeeding stages the size increases up to 17.9 microns at the fifteenth-day stage, and then decreases gradually to 13 microns by the thirty-fourth day, and to 10.6 microns by the seventy-fourth day stage. This gradual increase in size up to about the fifteenth-day period is due not only to the fact that the cells cease dividing, but also to the enormous amount of fat which they ingest, and which increases the volume of the cell.

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[11] Gluge, G.: Experimente über Encephalitis, Abhandlung z. Physiol. u. Path., No. 2, Jena, 1841, p. 13. (*vide* Essick 10.)

There was, of course, quite a difference in size in the cells of any one specimen, as, for instance, on the fifteenth day of repair, when cells were found to range between 10 microns and 42.7 microns (Fig. 10 shows a large cell), but this was an extreme case. The smallest cells, 7 microns, were noted on the second and tenth days, and the largest, 42.7 microns, on the fifteenth. A number of large cells were also found on the twenty-fourth day. The largest macrophages congregate in the region of the necrotic plug, and later occupy the space from which it has been removed. This is especially noticeable in the stages later than the fifteenth day.

By combining and averaging the size at the different stages it was found that the average long diameter of the macrophage, for the entire period from the second to the seventy-fourth day, inclusive, was 13.6 microns.

The shape of the macrophages, in the early stages, is very irregular. When found in crowded clumps they are polyhedral in form, and stellate outlines predominate in the looser groups. When lying free they are characteristically spherical or oval (Figs. 8 and 9). Rounded forms are the rule in the later stages (Fig. 17). Since they are ameboid it is probable that, especially in the earlier stages, they undergo frequent changes of form.

The cytoplasm is very voluminous in comparison with the nucleus. It varies in structure, of course, with the stage. It possesses the special property of being able to increase in size and to gather into itself materials from without which are stored within minute vacuoles. This evolution of the cytoplasm is quite rapid, so that the cells are of large size by the second day. This property gives to the cytoplasm a reticular appearance, especially in the sections prepared in the usual way, where the fat is dissolved out by the reagents. This reticulum becomes even more conspicuous with time, and so we find on the fifteenth day (Fig. 10), and later, typical examples of what have been termed "foam cells," the protoplasm being reduced to mere attenuated threads enmeshing the huge fat globules and aggregations of other foreign substances.

The material engulfed consists principally of dissolved protein and lipoids. The latter are particularly abundant in the brain, due to the large amount of myelin destroyed. Vacuoles, which probably represent fat, were found in all stages, including the seventy-fourth day. This substance was easily detected by the use of osmic acid, and by other methods. As early as the third day the cells contain as much fat for their size as they do in any later stage, although, of course, the cells are larger, as we have seen, in the later stages. The amount that each cell contains remains at this high level for two weeks or longer. The globules are of varying size, some being half the diameter of the cell, while others are very small. There is not much difference in their size from stage to stage, but, if anything, in the earlier stages the larger globules are proportionally a little more numerous. We will say more as to fat with the description of the fresh material. The osmic acid preparations show plainly the zones where fat-laden cells are most thickly congregated. Thus in S27-49 (three-day stage), transverse sections of the stab show a heavy ring of black cells (representing a special concentration of macrophages loaded with fat) just outside of the necrotic wall. This is especially clear cut in the transparent preparations. This ring of cells is not demonstrated with trypan blue.

Among the most conspicuous of the intracellular foreign materials is the blood pigment, which was found from the third to the seventy-fourth days, inclusive. It is easily recognized by the golden yellow appearance of the granule in the cytoplasm, and by appropriate methods of staining for iron. It is most abundant about the fifteenth day (Fig. 11). In the earlier stages (as at the sixth day) a few ingested red cells were found. It is an interesting point that there may be found closely associated in the same area of extravasated blood, macrophages whose cytoplasm is filled with golden yellow pigment, and others which do not contain a trace of this, these cells being otherwise apparently identical. Thus some of the cells appear to be especially developed for certain functions.

The blood pigment within the macrophage shows the familiar difference in tinctorial reaction with eosin, as compared with free red cells, pointing to a change having taken place in the hemoglobin, probably due to the activity of the phagocyte. Aggregations of extravasated red cells were frequently noted in the lesions, and were found as late as the thirty-fourth day of repair. The absorption of red cells in these aseptic injuries in rat brains seems to be slower than that of the red cells in the vicinity of abscesses in the brains of cats described by Essick.[10] It may be that here the infection hastened in some way the absorption of the red corpuscles.

In addition to materials apparently in a liquid form, the usual cell fragments, and even whole cells, were found engulfed by the macrophages. On the whole, it may be said that in the earlier stages, as from the second to the twelfth or fifteenth day, fat was the most conspicuous cell content, whereas later hemoglobin residue became more and more the dominant enclosure.

The nucleus of the macrophage is rounded or oval in form, and is situated characteristically eccentrically. It is well supplied with chromatin, and has usually a large nucleolus. As a rule, it is single, but not infrequently cells with two nuclei are found, suggesting an amitotic nuclear division (Macklin[12]). There was no constant variation in size of the nucleus with that of the cell as a whole; there was, too, no constant change in the chromatin material with advance in the repair process, nor did the size of the nucleus vary with the stage of repair. The average long diameter of the nucleus, as taken from stages of the second, twenty-fourth and sixty-second days, was 5.1 microns.

The method of multiplication of the macrophages is quite evidently by mitosis. Many dividing figures were found on the second day, and they were particularly abundant on the third day. They were found as late as the sixth day. No evidences of direct cell division were found. The presence of dye, fat, and other inclusions within the cytoplasm does not prevent division (as other workers have noted in similar cases), for we found many dividing cells which presented this condition.

In these aseptic cases but few polymorphonuclear leukocytes were found associated with the macrophages, as in the fracture cases (Macklin[2]) and, like these latter, they contained no dye granules.

Vital Staining of Macrophages.—The most interesting feature of the macrophages in connection with this investigation is their behavior toward trypan blue. The staining is notable for its short duration and for its weakness. The first macrophages which were found to contain dye granules were those of the pia-arachnoid in the vicinity of the lesion. In the sections these were first noted at the end of twenty-four hours, although some of the earlier stages examined with the binocular microscope disclosed them. They will be described in a special section to be devoted to the pia-arachnoid phagocytes.

In the macrophages of the brain lesion dye granules were first seen in the cells at the end of forty-eight hours. Apparently, as the cells develop phagocytic properties they take up the dye from the surrounding fluids. At this period the ingested dye was slight in amount, and it was necessary to search carefully in order to find it, although it was more conspicuous at this stage, and shortly after, than at any other. The coloration was best studied in thin sections cleared without counterstaining. At this stage many cells of identical morphologic type to those vitally stained were observed to contain no dye, and many also which had but very little. Indeed, the actual proportion of cells in which dye could be demonstrated, as compared with the unstained cells, was small. Cells with and without dye inclusions, but all evidently macrophages, were often mixed indiscriminately together in close association. Similarly, it will be recalled, we found macrophages filled with blood pigment in close association with those containing none of it. Most of the vitally stained cells were present in the necrotic wall and intermediate zone, as we should expect, since most of the phagocytes are found here. In none of the cells was the dye content large in relation to the total volume of the cytoplasm (Fig. 8), and most of the space is taken up, even at this early stage, by fat. The dye and fat seem to occupy different parts of the cytoplasm, the dye granules lying in the interstices between the fat globules. Mitotic figures were numerous in these cells.

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[12] Macklin, C. C.: Amitosis in Cells Growing in Vitro, Biol. Bull. **30**: 445, 1916.

It is worthy of note that we find, farther out, in what we have termed the peripheral zone (even where the nerve tissue shows only slight damage) cells containing dye granules (Fig. 24). These are not associated with vessel walls or perivascular spaces, and are undoubtedly neuroglia cells. Except for their dye content they cannot be distinguished from the neuroglia cells farther out in the brain substance. It was impossible to stain them so as to demonstrate their specific tinctorial reactions as neuroglia, and at the same time preserve their dye granules, since the small amount of blue dye contained in them is totally obscured by neuroglia stains. It was easy, however, to study the general morphology and at the same time inspect the dye content in sections lightly washed with carmin. Hematoxylin and eosin preparations were, of course, used for comparison.

As one progressed nearer and nearer to the site of injury, more and more of these cells could be found, larger as they were encountered closer to the necrotic wall, until they finally occurred as free rounded or oval cells, lying in the intermediate zone. Their stimulation into phagocytic activity is probably due to their being situated in an area of inflammation. The amount of dye within these cells is so slight as to be quite insufficient of itself to account for even the meager amount of color that we find in the peripheral zone, in the gross specimens. This pale diffuse color, it will be remembered, is quite imperceptible in the sections, and is most apparent in the freshly fixed gross material.

This vital staining reaction on the part of the neuroglia cells, and the occurrence of a series of transitional forms leading from the neuroglia of the peripheral zone to the typical brain macrophages of the necrotized tissue, lead us to look on these cells as one source of the macrophages of the brain.

On the third day the vital staining of the macrophages is about the same as on the second (Fig. 9). It is probable that a little more dyestuff has been phagocytized, but, also, more of the débris has been taken up, so that the dye content is not increased relative to the total volume of the cytoplasm. There are at this stage also many phagocytes, filled with fat and blood pigment, which contain no dye; indeed most of the cells do not contain it. Large numbers of mitotic figures appear here also, and some of the dividing cells enclose dye within their cytoplasm.

On the fourth day there is but little dye to be found in the cells and, in comparison to the amount of other material phagocytized, it is insignificant. It grows gradually less, and by the sixth day it is almost impossible to find in these free phagocytes any intracellular dye. Even when found the granules are small and few. After this no vitally staining phagocytes were found in S27 except a very few in the limiting membrane and its continuations, and in the brain tissue immediately surrounding the lesion. Scattered cells in these parts were found on the seventh and eighth days of repair. They were often in the perivascular sheaths. While plainly stained they were never conspicuous on account of their dye content. In form they were typically elongated and showed processes.

Briefly, then, we may say that in the lesion itself cells containing appreciable quantities of dye were found in the hot stab cases on the second and third days of repair, while on the fourth day the amount was very slight and on the sixth day it was hardly demonstrable.

Under the conditions of our experiments, then, it is obvious that the reaction of the macrophages toward the vital dye differs widely with the stage of repair. With advance in age and stage of development they fill up more and more with ingested material from the injured area, and their phagocytic powers become gradually fatigued. Hence it was to be expected that great variation in the results obtained would occur when we exposed cells to a standard treatment with the dye the cytoplasm of which was already occupied by varying amounts of foreign material. In stages later than forty-eight hours the fat and other débris have been in contact with the phagocytes longer than the dye has, and this discrepancy becomes more and more magnified with the passing of time. A cell of the fifth day, for instance, cannot be expected to give the same reaction toward the dye as one of the second day, for the latter is much younger, and has been exposed to the dye for the entire period of its hyperactivity owing to the stimulation of the trauma, whereas the five-day cell has been at work three days before the dye was exhibited to it, and by this time it has become filled with other material. It is not unlikely that it has become especially sensitive to the fat and protein débris which it encounters in its environment, and has become correspondingly insensitive toward other materials; and hence the dye finds it unresponsive. Indeed, the older the cell grows the less is it likely to take up the dye.

The same conditions obtain with phagocytes in tissue other than brain, for here, too, the staining power of the phagocytes gradually diminishes, as Macklin[2] and others have shown. In these tissues, however, since fat is not so abundant, it does not so markedly preponderate over the dye in the cell's cystoplasm.

It would appear that, even after the macrophàges, under the conditions of our experiments, failed to take up the dye from the fluids of their environment, they were still capable of phagocytosis, for we note a gradual increase of blood pigment within the phagocytes for some time after the sixth day. There is also reason for thinking that the cells are still taking up fat after the sixth day, for free fat is encountered in the lesion for some time after that (as is easily demonstrated by the use of osmic acid), and some of the cells gained appreciably in size. Moreover, the occurrence of diffusely stained débris up to the tenth day, or even later, shows that there is work then for the phagocytes. It may be that if the dye had been given in more massive doses, or had been exhibited over a longer period, that these cells could have been made to take it up. We did not expose these older cells to a treatment with the dye for a period longer than forty-eight hours. We did, however, expose cells, in several cases, from their beginning, to a prolonged treatment with the dye, as the following account will show.

Effect of Prolonged Staining.—The dye was administered at the time of operation (the usual hot stab) and twenty-four hours later, in the usual doses (15 c.c. in S27-47), but instead of killing the animals at the end of the forty-eight-hour staining period, as in the other series, we allowed them to live for varying lengths of time, so that the macrophages were exposed to the dye from their beginning to the time the animal was sacrificed. There was quite a high mortality, and the prolonged action of the dye was markedly toxic. Two cases survived long enough for use—one to the eleventh (S27-45) and one to the fifteenth day (S27-47).

The animal killed fifteen days after the operation, which thus had endured fifteen days of staining, showed all of its tissues much more distinctly colored than those of any other case. The presence of dye in the tissues points to the occurrence of some, at least, in the circulation, up to the time of death, for the experience of other workers has shown that in stained animals, which are allowed to survive, the dye diffuses out of the tissues until they are decolorized.

There was a diffuse bluish stain over the entire cortex, and the wound area was intensely blue, in contrast to that of fifteen days in Series 27. The coagulum was almost all present, whereas it should be largely resolved at this stage. It was stained at least as intensely as it was in any stage in Series 27. In it there were still myriads of the same dense blue granules which were described only for the early stages of S27. These facts all point to an inhibition of the absorptive processes due to the toxic action of the dye. This may be exercised on the enzymes which liquefy the débris, or on the phagocytes which play such an important part in absorption.

When we examine the slides we find that the lesion is filled with very large macrophages. They were at least as numerous as in any case we had, and stained very markedly with the dye, in contrast to the cells of the corresponding stage of S27, which contained no dye. Not only is there very much more dye within the cells than in any of the cases of S27, but the cells are practically all stained. The dye is distributed throughout the cytoplasm, between the abundant fat and blood pigment inclusions, which are present. The cells are plainly alive, for the nuclei are unstained and they are quite evidently discharging their phagocytic functions; however, the slow absorption of the débris in these cases points, perhaps, to a slackness in activity of the phagocytes, due to intoxication by the dyestuff, so that there is some weight in the contention that these phagocytes are physiologically subnormal. In no case was the volume of the dye within the cell such as to lend any support to the argument that the cells failed to take up débris because all their potential space was occupied by dyestuff.

The only evidence of progress in healing which this case shows over the earlier stages of S27 is that the membrane has started to grow down from the pia-arachnoid to line the edges of the wound. It is of interest that many of the cells constituting this membrane are filled with blue granules. They do not resemble the fibroblasts about them, which contain no dye, and are probably to be looked on as ameboid arachnoid cells.

Comparison of Brain Macrophages with Those in Other Inflamed Tissues.—One of the outstanding features of the macrophages of the lesions in S27 is the marked difference in response to the vital dye, trypan blue, which they show as compared with the macrophages of other injured tissues. If we contrast the phagocytes from aseptic inflammatory areas of liver, spleen and lymph gland, which Tschaschin[1] found, and shows in his figures, or those from the damaged tissue around fractured bones, which Macklin[2] describes, we are struck with the enormous difference in their vital staining in comparison with the brain macrophages. In Macklin's fracture cases the animals were subjected to approximately the same treatment with the vital dye as were these brain-wound cases of S27. At the three-day stage, and even before, in the fracture series, Macklin found the area about the injured bone quite blue with these cells, which he terms "extraosseous" macrophages (in contrast to the "intraosseous" macrophages which later inhabit the spaces of the resolving callus). His slides (Fig. 7) show enormous numbers of them, many being literally stuffed with the dye, whereas the cells from the brain wound cases of the same period contain but little dye by comparison. This difference is strikingly brought out by comparing slides, cleared without counterstaining, of brain wounds and fractured areas, of the three-day stage. In the latter the field appears very distinctly blue, whereas in the former the coloration is very weak indeed. The contrast is even more obvious in the slightly later stages; compare, for instance, the six-day stage of the fracture cases with that of the brain cases. In the latter, as we have seen, the dye content of the cells is practically nothing, whereas in the fracture cases (except in the young scar) there is usually an enormous amount of the dye within the cells. Then, too, stained cells in the fracture cases are found later than in the brain specimens.

Indeed so little dye is taken up by these brain phagocytes in contrast to the mononuclear phagocytes present in aseptic inflammation in other tissues as to call urgently for explanation. We can hardly suppose that the dye was any more accessible to the phagocytes of the fracture cases than to those of the brain cases, for in the brain there was much dye within the dead tissue, and thus there must have been an abundance of it available for the phagocytes of the vicinity. The soaking up of the dye by this dead tissue could not be looked on as depriving the phagocytic cells of their quota, for the entire amount of dye taken up by the dead tissue must have been insignificant as compared with the total amount of available dye in the circulation.

The assumption of an earlier or greater fatigue on the part of the brain cells, as contrasted with those of other inflammatory areas, to account for the greater amount of dye in the latter cells, has no basis of support, since in both cases the cells seem to be stimulated to the height of their phagocytic activities, and there is no reason for assuming that they would tire earlier in one case than in the other. It is possible that there is a greater volumetric proportion of débris to dye in the brain cases than in the lesions of other tissues, but this is difficult to prove. The fact that the cell goes on ingesting other materials after the sixth day (which is practically the last appearance of the dye), proves that the failure to take up more dye after the sixth day is not due to lack of potential cytoplasmic space. Nor does the attempted explanation based on difference in origin of the cells help us, it being claimed that the ectodermal neuroglia cells of the brain lesions do not show the same type of phagocytic activity as the phagocytes of other tissues, in that these neurogliogenous phagocytes are more specialized to ingest fat. It is difficult to prove this assumption, and, too, we do not know how large a proportion of the phagocytes come from the neuroglia cells; moreover, we do know that many of the brain phagocytes come from cells of mesodermal origin, such as the endothelial cells, so that this explanation cannot be looked on as of importance.

We might assume that the dye granule is produced by some sort of union—physical or chemical—between the aggregations of material already in the cell and the dye, and to argue from this that the cell which contains the greatest amount of such material will appear most strongly stained. Further, we might say, that the reason we get more staining in the case of the macrophages of the fractures is because these cells have more material (probably of protein origin) within them which takes up the dye than have the cells of the brain tissue, which are largely filled with fat, a substance in which the dye does not dissolve, and with which it does not combine. However, Evans and Schulemann[13] and others are opposed to the view that the dye granule is produced by a staining of material preexisting in the cell, and find that the granules of the macrophage arise by concentration of the ultramicrons of the dye within minute vacuoles. Although Shipley[4] believes that the dye and other materials may become mixed together within the vacuole, he feels that this is to be looked on simply as a mechanical mixture, and he holds that the substances within the vacuole have been impounded there probably by virtue of the peculiar mechanism of the cell, working, it appears, through an adsorption of the various molecules on the vacuole wall as a preliminary to their penetration into its interior. Foreign material already within the cell vacuole may be presumed to be inert so far as exerting any attraction for the dye, or as acting to incarcerate it in any way, is concerned. Even assuming that the granules in the early stages were engendered by the concentration of dye in preexisting intracellular ingesta, it is difficult to explain why this material should so suddenly diminish in amount after the second and third days, while such substances as fat and blood pigment continue to be taken in.

Perhaps the most plausible explanation depends on the important chemical differences which these tissues show, in that there is much more fat in the brain (due to myelin breakdown) than in the fractured bone, and it may be that the phagocytes of the brain become specially developed to ingest this fat, whereas those of the fractured bone cases become specialized to ingest protein. However, this difference is only relative, for we must assume that there is a considerable amount of broken-down protein in the brain tissues, and also a goodly amount of fat in the fracture cases. Indeed in the fracture cases, notably about the sixth day, cells with heavy loads of lipoid and but little dye were fairly frequent. On the whole, however, it is incontestable that there is more protein breakdown in the fracture cases, especially where muscle is damaged; and, vice versa, more free fat in the brain cases. We may assume, then, that the macrophages of brain tissue become especially developed to take up fat, because of the relatively enormous amount of it to which the cells are exposed, whereas, for the same reason, a similar preponderance of the products of proteolysis in the case of the fractures determines the larger amount of these materials which the cells take up. Finally, it must be assumed that the dye is more like the dissolved protein physically than it is like fat, and that hence the cells which are specialized to take up protein products ingest dye more readily than do those which are developed to take up fat.

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[13] Evans, H. M., and Schulemann, Werner: Ueber Natur und Genese der durch saure Farbstoffe entstehenden Vitalfärbungsgranula, Fol. Haematol. **19**:207, 1914.

These brain macrophages, too, are distinctive in that there were probably more cells without dye, even in the second and third day stages of S27, than with it, the converse being true of the fracture cases. This paucity of stained cells cannot be due to any inherent inability on the part of certain of the cells to take up dye, for in the prolonged staining cases, where the cells were given dye for an extended period from their beginning, they practically all stained. Thus the unstained cells are not essentially different from the stained. Fragmented forms, like those found in scarring areas in the fracture cases, were not noted.

There is still another important difference which presents itself when we compare these brain phagocytes with those found in wounds of other tissues. Tschaschin[1] found in his aseptic inflammatory tissue of spleen, lymph gland and liver a host of transitional cells, ranging from a small cell, looking like a lymphocyte, to the large "polyblast," or macrophage, and Maximow had previously noted the same condition. In Macklin's fracture cases an identical transition is very evident. Indeed it would seem probable that a great number of these macrophages are developed from small lymphocytoid cells, coming, apparently, largely from the blood stream. When we examine the brain cases, however, we find hardly a trace of such transitional cells, and it may be said with confidence that, if lymphocytoid cells contribute at all to the macrophages here, they do so in such small numbers as to be negligible.

In attempting to account for this condition it may be assumed that in the case of wounded brain there is not the same peremptory call for assistance from premacrophages from outlying regions that there is in the case of wounded muscle, liver, spleen, etc. This call, probably, is due to the diffusion into the blood stream of small quantities of split proteins.

Not only were there enormous numbers of transitional cells among the extraosseous macrophages of the fracture cases, but there were few mitoses, whereas in the brain cases there were very many mitoses. This points to a derivation of the cells of the peri-osseous tissue from a source outside the lesion, in contrast to the brain wounds, where the cells seemed to be derived from the brain tissue itself.

Tissue Transplants.—We thought that, by inserting into the brain wounds small quantities of other tissues it might perhaps be possible to attract thither premacrophages, and that these might even develop into macrophages of a more trypanophilic type, so bringing about a picture similar to that in aseptic inflammation of other parts. Accordingly, we planned, and carried out, several additional experiments. In three cases (Series 29) we inserted a piece of temporal muscle into a hole in the brain made by cutting, without burning, and examined at the second-day, sixth-day and tenth-day stages. Resolving muscle tissue, it was found in the fracture cases, was especially noteworthy because of the occurrence in it of enormous numbers of phagocytes loaded with dye, and also of myriads of transitional cells. In two cases (S30) we inserted similar pieces of temporal muscle into a hole burnt with a hot needle, as in S27. In S32 the hole in the brain made by cutting was filled with liver; two cases were prepared, and examined at the second-day and sixth-day stages. In two cases of S33 similar wounds were filled with spleen tissue and both examined on the second day. In all of these cases the tissues were sterile and fresh, and were taken from similar rats, killed at the time of operation.

In none of these cases were there found in the brain lesion any transitional cells, nor was there any increase in the number of macrophages. It cannot be denied that products from the breakdown of these tissues diffused into the surrounding fluids, but there is no evidence that they attracted phagocytes from the blood stream. There was some delay in healing, in these cases, due to the greater amount of tissue to be absorbed. The phagocytes of the surrounding brain did not develop any greater avidity for the dyestuff, and continued to show the meager staining which characterized the cells of S27. In size (by actual measurement), the phagocytes of these special cases did not show any material deviation from that of the cells of S27 Their position in the lesion was also similar, and they did not show any more mitotic figures. These experiments were, therefore, negative in giving us any explanation of the differences in staining intensity of the cells under discussion, or in enlightening us as to the origin of the brain phagocytes.

Abscess in Lesion.—In contrast to the negative results obtained in the foregoing experiments, were the observations made on a small abscess which occurred by accident in one of the cases operated on as in Series 27, and which thus had to be omitted from the aseptic cases. It gave most interesting information as to the reaction of the macrophages toward the dye, under such conditions. The animal (S27-41) was killed at the twelfth day of repair.

In the gross the specimen was characterized by the dense blue color of the lesion (in contrast to the regular members of S27 of this stage, where the dye had almost, if not absolutely, faded), showing that the removal of débris has been somewhat delayed. The upper surface of the brain presented a dark blue plug, filling the defect, surrounded by a narrow, paler zone. In the hemisection this mass appeared particularly densely stained and projected above the brain contour, occupying the upper end of the defect like a cork in a bottle. This was the abscess. Lower down the blue staining extended almost to the end of the stab. There was practically no blue in the walls of the defect.

Sections through the stab (Fig. 22) show the small abscess occupying the aforementioned projecting plug. It consists of an aggregation of pus cells, enclosed by a well marked capsule of macrophages, which stain densely blue, and give to this tissue its deep color. Outside of this macrophagic wall is a fibrous zone, representing the membrane, described as effecting the delimitation of the field. This fibrous tissue is directly continuous above with the pia-arachnoid, which here is much thickened. Surrounding this membrane is the brain tissue.

Thick, clear and uncounterstained sections present a striking picture (Fig. 7). The macrophage zone stands out as a dense blue ring, the stain of which can be accounted for by the granules in these phagocytes. Within it the aggregation of polymorphonuclears is stained a pale diffuse blue, but no dye granules are demonstrable in these cells. Here and there among the polymorphonuclears, especially in the periphery of the collection, are macrophages which have wandered in. There are, of course, polymorphonuclears to be found in the macrophagic zone, but these become outwardly less and less in number. No dye granules were found in them. No bacteria were demonstrated, and the septic process appeared to be very subacute as compared with that obtaining in the experimental abscesses in cat brains described by Essick.[10]

These abscess-wall macrophages are large, and, when their contour can be discerned, they are more oval and elongated than those of the brain. From their close relation to the pia-arachnoid they seem to be developed from it.

An area from the section from which Figure 22 was photographed is seen in Figure 25. This shows the various layers of the abscess, the aggregation of pus cells in the center, the surrounding macrophage capsule, the enveloping fibrous tissue, and finally, sharply marked off from it in places, the brain tissue. The latter shows, as in the aseptic cases, a much changed structure in the zone bordering the membrane, with loss of ganglion cells and a large meshed intercellular reticulum. A small group of dye containing macrophages, taken from this field, is shown at a higher magnification in Figure 23. It is easily possible to find macrophages here which do not contain dye, yet the majority do so. In Figure 23 the cytoplasm is shown of an indefinite contour, and, indeed, the impression is given of a syncytium.

Lower down in the stab the débris is being absorbed, as in S27, but somewhat more slowly, due to the superposed infection. The usual numbers of large, fat-laden macrophages are seen. They contain no dye, as in all the S27 cases after the sixth day, and thus stand in sharp contrast to the macrophages of the abscess wall. Indeed, the latter contain much more dye than did any of the brain macrophages, even in their most marked stages. This is strikingly shown when this specimen is compared with cleared slides from the second and third day stages of S27. The macrophages of the abscess wall, indeed, resemble, in their avidity for the vital dye, much more closely those found in the fracture cases. Their specialization to ingest the toxic products which have diffused out from the abscess has doubtless fitted them for the ingestion of the vital dye, and in this respect they are physiologically more like the macrophages of inflamed nonnervous tissue than they are like the macrophages of the brain lesions.

The difference which the macrophages of the abscess wall show, as compared with the neighboring polymorphonuclear leukocytes, in respect to their behavior toward the vital dye, is most striking, and speaks strongly in favor of the physiologic distinctness of these two cell types. The amount of dye contained in the mass of leukocytes is so slight as to be hardly observable, even in very thick sections cleared without counterstaining; indeed, at first glance at such a section it appears as though the contents had fallen out of the abscess cavity, and that we were seeing only the bright blue wall. The leukocytes of the abscess wall, and beyond it, show the same condition. This meager diffuse staining is less than that which one usually finds in moribund tissue which has been subjected to the action of trypan blue intra vitam. In showing an entire absence of dye granules these leukocytes are quite similar to the few polymorphonuclears which were found in the aseptic brain injuries and in the area of inflammation in Macklin's fracture cases. It may be, as would be suggested by the work of Downey,[14] that the polymorphonuclears did take up the dye in quantity, holding it in granular form, during the first hour or so, and later allowed it to diffuse out. To settle this point we would have to examine the abscess very soon after staining with trypan blue. But even admitting this to have occurred, would not such an early ingress of the dye into the polymorphonuclear, followed by its almost immediate egress therefrom, constitute a most important physiologic distinction as compared with the macrophage, which not only is slower in taking up the dye (according to Downey) but retains it in granular form for a very much longer time? Is it not probable, too, that this very decided functional difference in reaction to trypan blue and similar substances is to be accounted for by the peculiarities of structure which these cells show—we might even say largely by the equipment of the macrophages with their innumerable vacuoles, analogous, as Shipley[4] has pointed out, to the food vacuoles of the protozoa? We would emphasize as important the fact that the two cell types show such an enormous difference in respect to their reaction to trypan blue under the conditions of our experiments—a difference in keeping with the other evidences of functional separateness which these cells show, such as the tremendous enlargement which the macrophage undergoes in consequence of its enormous intake of foreign material, in contrast to the practical absence of hypertrophy on the part of the polymorphonuclear cell. The attempt to account for such marked disparity in behavior on the ground of "availability of the dye," as Downey[14] suggests, seems inadequate, to say the least, when one considers that vitally stained macrophages are often found scattered among polymorphonuclears which contain no dye granules in an intimacy of juxtaposition that leaves no reason for concluding that the dye is any more available to the one cell type than to the other.

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[14] Downey, H.: Further Studies on the Reactions of Blood- and Tissue-Cells to Acid Colloidal Dyes, Anat. Record **15**:103, 1918. Reactions of Blood and Tissue Cells to Acid Colloidal Dyes Under Experimental Conditions, Anat. Rec. **12**:429, 1917.

While it is undoubtedly true, as Downey[14] has repeatedly shown, that dye aggregations can be made to occur in polymorphonuclear leukocytes by subjecting them to very high concentrations of dye and unfavorable environment, as within a doubly ligated vein, or in the stagnated blood of an animal that died some time before the material was taken from it for examination, yet this method can hardly be looked on as tending to bring out the distinctive characters of these cells, as contrasted with the macrophages. Rather it is likely to reduce them to a common dead level, due to the toxic action of the dye (an action which Downey[14] admits—p. 123), when exhibited in such excessive amounts; and in considering Downey's leukocytes with their stained granules, one cannot help recalling the experiment of Evans and Schulemann,[15] of which the substance is contained in their quota-tion (p. 10): "When we tap the cover slip over leukocytes swimming in trypan-blue, mechanical injury to the immediate subjacent cells invites instant entry of the dye."

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[15] Evans, H. M., and Schulemann, Werner: The Action of Vital Stains Belonging to the Benzidine Group, Science, N. S. **39**:443, 1914.

Cold Stabs.—As a comparison with the hot stabs a number of rats were operated on by simply stabbing the brain with a sterile cold needle, the rest of the technic being similar to that of S27. The tissue damage, of course, was much less, and thus there was but little diffuse staining. Macrophages of the same type and of similar size (by actual measurement) were present, but their numbers were less. In a given volume of damaged tissue, however, they seemed to be as numerous as in the hot stabs. Their vital staining was in all respects similar to that of the phagocytes of the hot stabs.

Fresh Material.—In a number of cases (not included in the table of material) we studied these brain macrophages in the fresh tissue, in some cases from vitally stained animals, and in others from animals simply operated on without staining. The operative and staining technic was the same as that in S27. Our interest centered principally around the dye and fat content. Most of the cases were examined on the third day, when the phagocytes are at the height of their activity. The latest stage examined was the eighth day. Immediately after being killed by bleeding, under ether anesthesia, the brains were dissected out and at once placed in normal Locke's solution at 37 C. Bits of tissue from the wound area were teased out on a slide and kept on the warm stage during the examination. In those animals which had received the vital dye the macrophages were plainly evident by reason of their dye content. They seemed to contain no more than in the fixed preparations. Sudan III was then added to the preparation and the cells appeared filled with large orange colored droplets. Many similar droplets were floating about free in the surrounding fluid. The treatment with sudan III, however, tended to decolorize the dye. This is doubtless due to its alkalinity. The same brilliant staining with sudan III was, of course, obtained in the animals which had received no dye. Some of the teased preparations gave most interesting results, for it was easy to separate the long slender capillaries which are so prominent during the early stages of repair. Lining these tiny vessels were to be found numerous protuberances, which stained with sudan III, and were quite evidently macrophages attached to their walls. The walls themselves contained globules of stained fat, and some of these were even present in the lumen, suggesting strongly a passage of the fat into the vessels—a process in which the macrophages may play a part, for they are ameboid, and it is not difficult to think of them as gathering their load of fat and carrying it to the vessels, into which they deliver it. A somewhat analogous relationship has been described by the Clarks[16] for leukocytes and lymphatic sprouts in the transparent tails of tadpoles, which suggests the well-known hypothetical action of the leukocyte in fat absorption from the intestine. Irrespective of such a speculation as to the action of the macrophage in fat absorption from brain lesions, the taking up of fats and other materials from such areas by the blood vessels appears to be pretty certain, for the subarachnoid spaces do not seem to receive much of these waste products, and the ventricular system apparently is not, in these aseptic cases, an important pathway for the exit of these substances. The important rôle which the blood vessels play in the removal of exudate has been recognized for some time. Recently Shipley and Cunningham,[17] in their valuable work on the taking up and removal of fine particulate matter and colloidal and molecular solutions from the peritoneal cavity, have demonstrated that this function is carried on largely by the blood vessels of the omentum. Downey,[14] too, has found evidence that the blood vessels of the subcutaneous connective tissues may drain away foreign materials. Such an action on the part of the young vascular sprouts of brain lesions is only what we might expect, and it seems probable that the increased staining with trypan blue which we noted in the larger vessels in the region of the injury may be part of the same functional adaptation. Since the process of fat absorption extends over quite a long period it is not likely that the fat content of the blood is appreciably raised above normal at any time. It is interesting to note that Coen,[7] at the second day of repair of similar brain injuries, noted fat droplets in what he terms lymph vessels, and also in perivascular spaces.

Frozen sections stained with sudan III gave similar pictures to those from fresh teased material, though not so striking, for not such long stretches of the vessels could be isolated. Many of these cells are undoubtedly modified endothelium. Of course, a great many phagocytes were detached from the vessels—indeed it is probable that most of them were. In preparations stained with osmic acid the same relationship of fat-containing phagocytes to the vessels was obtained. Nile blue sulphate was used, both with the fresh tissues and frozen sections, but the results obtained were unsatisfactory, for this dye was found not to be specific for fat, and so did not serve to differentiate it as did the sudan III.

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[16] Clark, Eliot R., and Clark, Eleanor Linton: A Study of the Reaction of Lymphatic Endothelium and of Leukocytes, in the Tadpole's Tail, Toward Injected Fat, Am. J. of Anat. **21**: 421, 1917.

[17] Shipley, P. G., and Cunningham, R. S.: Studies on Absorption from Serous Cavities. I. The Omentum as a Factor in Absorption from the Peritoneal Cavity, Am. J. Physiol. **40**:75, 1916. II. The Histology of Blood and Lymphatic Vessels During the passage of Foreign Fluids Through Their Walls, Anat. Rec. **11**:181, 1916.

Pia-Arachnoid Cells.—We have referred, in the description of the gross specimens, and again when speaking of the staining of macrophages, to certain of the cells of the pia-arachnoid membrane which, in the vicinity of the lesion, show a propensity for dye ingestion and storage. They are found in the infoldings of pia near the lesion, as well as on the exposed surfaces. We have described them as occurring almost constantly in S27, from the sixth hour to the sixth day, and from that time intermittently up to the twenty-fourth day. They were present in a depressed fracture case on the fourth day. In the matter of dye ingestion these cells were somewhat more precocious than the brain phagocytes; they also differ in taking up more dye, and so appear more conspicuously stained than do the "Körnchenzellen." In shape they show a tendency toward oval and elongated forms. Dye was often found clumped in grayish-blue masses half as large as the cell, and, in considering the cells of the vicinity of the lesion one cannot escape the impression, especially in the stages immediately following the injury, that the cells are seriously damaged, and so absorb the dye, rather than that they take it up by reason of their phagocytic activities. The latter are undoubtedly operative in the cells more remotely situated from the lesion, however, and in the later stages, for typical dye granules are found in them. Appropriate stains demonstrated the presence of fat globules in these pia-arachnoid cells. In the microscopic sections dye was first found at the end of one day.

Although it seems probable that some of the diffusible products of inflammation find their way into the subarachnoid space, especially in the early stages, yet the rarity of occurrence of dye-reacting cells in the membranes at any distance from the lesion points, in our mind, to but a very slight contamination of this interval. Doubtless the function of the cells here is to ingest any toxic materials which may enter. The cells of the pia proper show this reaction, and dura cells in the wound precinct manifest the same increase in dye ingestion. To them a similar function may be ascribed.

Choroid Plexus.—We have mentioned, in the description of the gross material, that the choroid plexus, from the first hour to the sixth day, was more intensely stained on the side of the lesion than on the opposite side. Normally, as is well known, the choroid plexus shows, in animals vitally stained with trypan blue, a distinct coloration. This is to be found in certain "secretory granules" of the choroidal ependyma (Evans and Schulemann[18]) but especially in the bodies of the macrophages, a number of which are always found in the plexus. It is in these cells that most of the dye is stored.

It would appear that, in the early stages, say the first twenty-four hours, the ipsilateral plexus shows an increased staining on account of its having been injured. However, it is noteworthy that the sections from the injured side soon show in the regular macrophages (first, second and third day stages) an increase in dye content (Fig. 28) as compared with those of the contralateral side, and it seems evident that these cells play a part in ingesting the excreta of the wound area. They did not show this increase after the fourth day. They contained fat globules in abundance. When the ventricle is penetrated, of course, the reaction of these cells is most marked, and much foreign material finds its way into the ventricular system. Even when the lesion does not involve the ventricle, but is situated close to it, we feel that some waste material may diffuse into it. We have, in the osmic preparations, found numerous globules of fat in the lateral ventricle, and it would seem that this space may become a drainage pathway for exudate arising in the adjacent parts of the cerebral hemisphere. However, the amount of material entering the unwounded ventricle, in our experience, was slight. Interesting in this connection are the observations of Essick,[10] who has shown that infections of the brain spread without difficulty from the lesion into the ventricle, and one gathers from his account that the ventricular system is more vulnerable, in this respect, than is the subarachnoid space.

The macrophages of the pia-arachnoid and of the choroid plexus must, therefore, be regarded as cooperating with those developing in the brain substance in dealing with diffusible effluxions from the inflammation focus.

In the healing of the wounds of skin, skull and dura, macrophages play a similar part, early rallying to the field and showing marked trypanophilic tendencies, as Macklin[2] has shown.

SUMMARY

The general features of the healing of aseptic lesions of the brain as brought out in this investigation may be briefly summarized. The defunct tissue is rapidly absorbed, and this is accomplished by a temporary cellular mechanism, made up of young blood vessels, phagocytes and connective tissue——in fact, a granulation tissue. This, in turn, gives place to a rather light scar. There is noted very early a separation of the irreparably damaged tissue from that reparably damaged — a separation at first indicated by a zone of ameboid mononuclear phagocytes, and later accomplished by a membrane of connective tissue which is continuous above with the leptomeninges, and is joined by strands of connective tissue with the reticulum which comes to occupy the defect. Repair of the brain tissue immediately surrounding this isolating membrane is not complete, and its essential composition, although not altogether destroyed, is profoundly changed. There is often an apparently insoluble residue of tissue which persists in the defect for two months or more; this, however, is very scanty. Red cell absorption was rather slow in these cases. An attempt is made to obliterate the defect, but this is often imperfect.

When foreign tissue was transplanted into the lesions there was some delay in healing. Delay was also noted when the exposure of the animal to trypan blue was prolonged.

Fully developed macrophages were present in the lesion in all stages from the second to the seventy-fourth day. Their period of most intense activity appeared to be from the second to about the sixth day, or shortly after. Though their sizes showed great variation, ranging from 7 microns to 42.7 microns, the average long diameter was found to be 13.6 microns.

The wound area, caused by stabbing with a hot needle, in the brains of rats into whom trypan blue has been introduced two days before death, presents a characteristic picture. As was to be expected, the dead tissue (both that which was homogeneously coagulated by the heat and the cells which still retain something of their morphology) is diffusely and deeply stained blue. Often the cells take up more dye than the intercellular material. This staining lasted from before the first hour to about the tenth day. It gradually became faint, as the stainable substance diffused out. Similar staining was found in cerebral lesions produced by other methods.

In the coagulated tissue in the early stages (six-hour to three-day) there were found granules which looked like the "dye granules" found in the cytoplasm of the macrophages. They are probably stained masses of protein decomposition products which have absorbed and concentrated the dye. They were also found in the coagulum at the eleventh and fifteenth days, where staining was of long duration.

Around the lesion in the early stages was an area of pale blue, probably the site of an inflammatory edema. This also occurred in concussion and depressed fracture cases, and in meningeal infection.

The superficial blood vessels of the injured side were more heavily stained than those of the other—perhaps denoting an increased permeability. In the inflamed tissue vitally stained phagocytes were found lying along the blood vessels and even composing part of their walls.

Vitally stained mononuclear cells appeared in the inflamed region at the end of the second day and disappeared by the sixth day, or shortly thereafter. They contained much lipoid material. The fat droplets and dye granules seem to occupy separate protoplasmic compartments. Blood pigment is found in cytoplasm also, being most abundant about the fifteenth day. Rarely are entire corpuscles found within the cells. As compared with macrophages occurring in wounds of other tissues these cells were remarkable for the small amount of dye they took up, and the short duration of their staining, when exposed to the dye for forty-eight hours. Then, too, in the brain cases many phagocytes contained no dye, whereas in the fracture cases practically all contained dye. This marked difference in response of brain macrophages toward trypan blue as compared with those occurring in other tissues is in all probability due to the larger amount of fat in which they are situated. Apparently these brain macrophages display phagocytic activity toward fat and blood pigment long after they cease to respond to the dye when exhibited for a period of forty-eight hours. Similar phagocytes appeared in the pia-arachnoid membrane around the lesion. They took the dye a little more avidly and somewhat earlier than the macrophages in the brain tissue.

If animals operated on were allowed to live for from eleven to fifteen days after injection of the dye, the macrophages were quite deeply stained. Such prolonged staining, however, seemed toxic to the animals, as evidenced by the high mortality. It inhibited to a very marked degree the absorption of the waste material, and delayed healing.

The macrophages of the brain lesions differed also from similar cells occurring in other tissues in that no transitional cells from lymphocytes were found, and that they multiplied by mitosis at the site of injury. They were apparently recruited in part from the neuroglia, for hypertrophied neuroglia cells containing dye granules and other material were found in the area of inflammation. Some also arise from the endothelium of the blood vessels, for endothelial cells in the injured region became enlarged and filled with foreign material. They then behave like the other macrophages. By careful dissection of the fresh or formaldehyd-fixed specimens, capillaries lined by large fat endothelial phagocytes may easily be isolated. These may also be found in the sections. It is possible that the cells of the pia-arachnoid, growing down into the wound, and adventitia cells from the ingrowing blood vessels, add their quota to the phagocytes, as found by several workers.

In a small abscess of the brain the wall was composed mainly of large macrophages. These stained very brilliantly with trypan blue — much more so than the brain macrophages of the aseptic cases at any period. They resembled closely the macrophages about fractures. This abscess specimen was examined at the twelfth day — a stage when no dye was found in the ordinary brain macrophages. This circumstance suggests that the type of material absorbed may have something to do with the positive reaction of the macrophage toward the vital dye. No dye granules were found in the polymorphonuclear leukocytes of the abscess.

Introduction into the brain lesions of other tissue material, such as liver, spleen, muscle, etc., in an effort to attract macrophages similar to those of inflammatory lesions in other tissues (i. e., cells that would take up more dye, and exhibit developmental forms arising from lymphocyte-like cells) gave negative results.

The function of the macrophages apparently is to ingest the products of tissue decomposition. It appears that they subject these products to a form of digestion (Shipley[4]). It is even suggested that fat may be transported by the macrophages to minute blood vessels, through the walls of which the globules pass, to be finally carried off in the blood stream. In any event, the greater amount of absorption of waste materials from the wound site seems to take place via the blood stream, although a little doubtless diffuses into the adjoining subarachnoid space and lateral ventricle.

The macrophages are incorporated in the scar formed by the fibroblasts, although there is no evidence in this work that they actually proliferate to form that scar. Some were found in the defect more than two months after the operation.

Cells of the dura about the lesion became phagocytic, and responded to the dye to some extent. The macrophages of the choroid plexus became more responsive to the dye than normal during the first three or four days of the injury.

Failure of a cell to respond to the vital dye cannot be held to exclude it from the possession of phagocytic activity, since the same cell early in its history may take up the dye, and later fail to do so, while at the same time continuing to ingest fat, blood pigment and other substances. Again, in some cell groups, many of the macrophages may contain no dye (and yet presumably be phagocytic, as evidenced by their content of ingested material) while others, right beside them, contain dye in appreciable quantities. Whether or not a cell contains visible dye granules, however, depends largely on the amount of dye given and the duration of the staining, as the special cases of prolonged staining show. Thus vital staining should be used with discretion as a macrophage indicator, as it is not always an infallible criterion.

Cold stabs showed no essential difference as compared with the hot stabs except the much slighter extent of the injury.

In conclusion, we wish to express our thanks to Dr. Streeter, Director of the Carnegie Laboratory of Embryology, for laboratory facilities and assistance with the illustrations generously granted.

EXPLANATION OF PLATE 1

Fig. 1.—Crayon drawing of brain of white rat which has been stabbed with a red-hot needle, and killed twelve hours after operation. Trypan blue, 4 c.c. intraperitoneally in 1 per cent. aqueous solution forty-eight hours before death, with a second dose of 4 c.c. twenty-four hours before death; S27-23, twelve-hour stage. Note intense blue of central necrotized area, surrounded by a zone of paler blue. Superficial blood vessels of injured side stain more intensely than those on the other side. The staining of pineal body is normal. This brain was cut transversely along the stab, and the cut surface is shown in Figure 3. × 2; drawn by B. E. Stocking.

Fig. 2.—Crayon drawing of brain of rat. Similar operation and staining to that of brain shown in Figure 1, but drawn from the six-day stage of repair; S27-14. The intense blue staining of the necrotized tissue persists, but the surrounding paler zone has diminished in size and in intensity of color. The superficial blood vessels still show more intense staining on the injured side. A cross section of the stab, from a cleared slice of tissue, is shown in Figure 4, and cleared longitudinal sections of it are seen in Figures 5 and 6. × 2; drawn by B. E. Stocking.

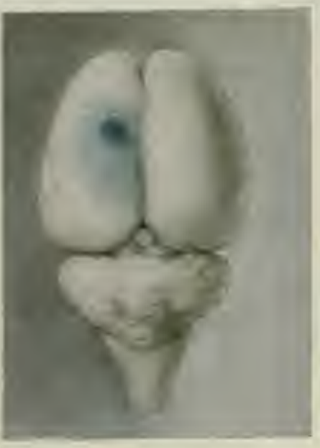
Fig. 3.—Crayon drawing of transverse section through the brain from which Figure 1 was drawn, showing a longitudinal section of the stab — S27-23; twelve-hour stage. The dense blue coagulated central plug is sharply marked off from the lighter blue zone surrounding it. × 2; drawn by E. Lovett.

Fig. 4.—Water-color-crayon drawing of thick slice cut transversely to the stab from the brain shown in Figure 2. Specimen cleared in oil of wintergreen and drawn under the binocular microscope—S27-14; six-day stage. Note the densely and diffusely stained coagulum occupying the center; around it is the paler intermediate zone. Next we see the necrotic wall, its inner margin appearing as a thin blue ring. The dye in this ring is partly in granule form, in the bodies of macrophages, and partly in a diffuse staining of the young connective tissue which is gathering here, and which represents the beginning of the membrane that will wall off the defect. The diffuse staining of the remains of the necrotized tissue also probably contributes to the blue color. Conspicuous in the surrounding inflamed tissue are the numerous blue granules, which represent dead nerve cells, as well as a few macrophages which contain vital dye. In the sections of the six-day stage there was very little intramacrophagic dye. The radiating blue lines are blood vessels, which appear so not only because their walls stain diffusely (more intensely than normal vessels) but also because their endothelial cells, and macrophages applied to their walls, contain dye. Similar vessels run upward toward the meninges, as seen in Figure 6, from the same specimen. There are some masses of extravasated blood. × 20; drawn by E. Lovett.

Fig. 5.—Water-color-crayon drawing of a slab, cleared in oil, cut transversely from the brain seen in Figure 2. This shows a stained lesion, as seen with the low power of the binocular—S27-14; six-day stage. The blue staining of the lesion is conspicuous, but the zones are not so well marked as in the uncleared specimens. The staining of the choroid plexus on the injured side is greater than that on the other side. The enclosed area is shown more highly magnified in Figure 6. A cross-section of this stab, made by placing the two halves together, is seen in Figure 4. × 4; drawn by E. Lovett.

Fig. 6.—Water-color-crayon drawing of a small area from Figure 5 (shown enclosed) as seen under the high power of the binocular — S27-14; six-day stage. The granular nature of the staining is due to dead ganglion and other cells, as well as to vitally stained macrophages. The amount of dye within the cells, as shown by sections, is slight. Again (as in Figure 4) we find blue lines, representing blood vessels. Here they are mainly running up to the meninges. × 44; drawn by E. Lovett.

PLATE 1



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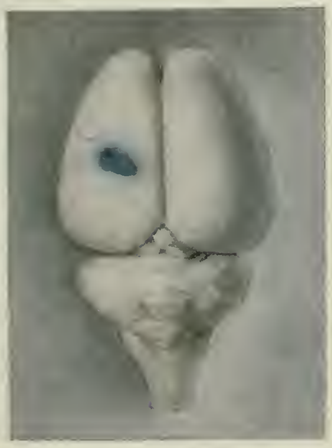

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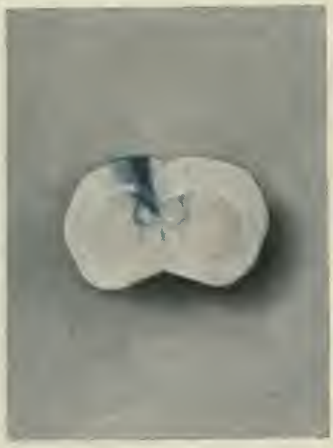

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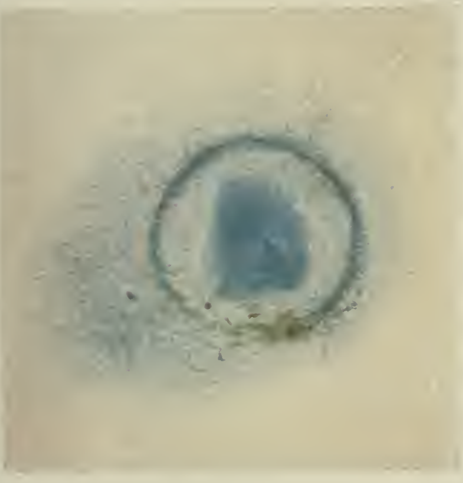

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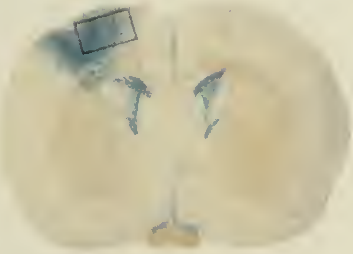

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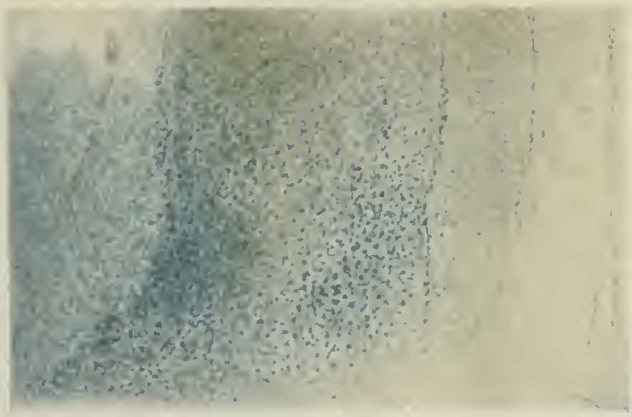

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EXPLANATION OF PLATE 2

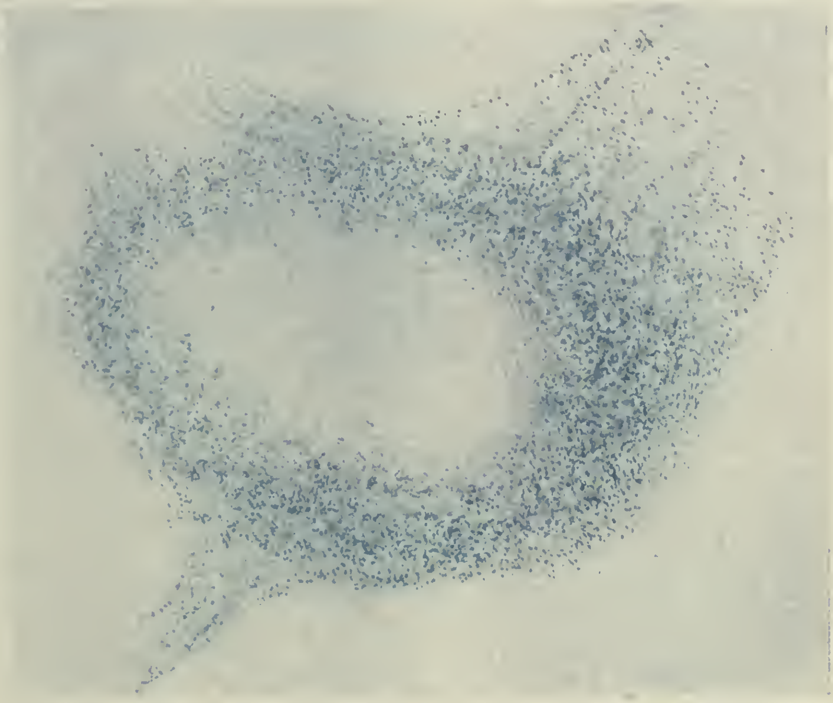
Fig. 7.—Water-color drawing of the abscess shown in Figure 22 as a photomicrograph—S27-41; twelve-day stage. The drawing was made from a thick section, simply cleared without counterstaining. The rather pale, diffuse blue area in the center is occupied by the pus cell accumulation, with the remains of the necrotized tissue. The conspicuous blue granular ring is due to the vital dye in the host of macrophages composing the envelope of the abscess. A section of the wall is seen in Figure 25, and a group of macrophages from it in Figure 23. × 80; drawn free-hand by S. E. Watson.

Fig. 8.—Water-color drawing of two macrophages found in the dead tissue of the two-day stage — S27-6-9; light counterstaining with carmin. Note the granules of trypan blue. These were particularly well stained cells, the vast majority containing less dye, or none at all. Figure 13 presents a photomicrograph of this stab, and in Figure 24 are seen macrophages from the peripheral region of the same specimen. Camera lucida with Leitz 2 mm. oil immersion lens and No. 6 compens; ocular; × 1500; drawn by M. T. M.

Fig. 9.—Water-color drawing of two macrophages from the edge of the coagulum at the three-day stage—S27-37-4; lightly counterstained with carmin. These, too, were selected because they contained more dye than most of the others. They are not quite so well stained as those of the two-day stage, shown in Figure 8. They were surrounded by red blood cells and other macrophages containing no trypan blue. Photomicrograph of this stab is seen in Figure 14. Camera lucida and oil immersion lens; × 1500; drawn by M. T. M.

Fig. 10.—Water-color drawing of a very large phagocyte found in the lesion at the fifteen-day stage—S27-18-8; hematoxylin and eosin stain. The cell contains no vital dye, and its cytoplasm is made up largely of a host of vacuoles. Its size may be estimated by the red blood cells around it. In Figure 15 a photomicrograph of the stab is seen. Camera lucida and oil immersion lens; × 1500; drawn by M. T. M.

Fig. 11.—Water-color drawing of similar cells from the lesion of the same specimen—S27-18-5; fifteen-day stage. Hematoxylin and eosin stain. These cells are particularly noteworthy for their content of blood pigment. Note size of red blood cells. Section of stab is seen in Figure 15. Camera lucida and oil immersion lens; × 1500; drawn by M. T. M.



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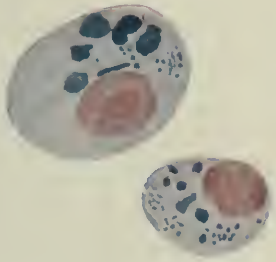

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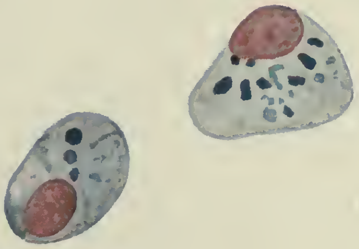

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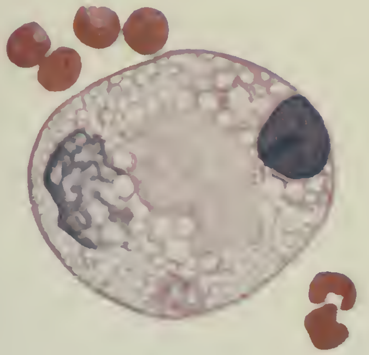

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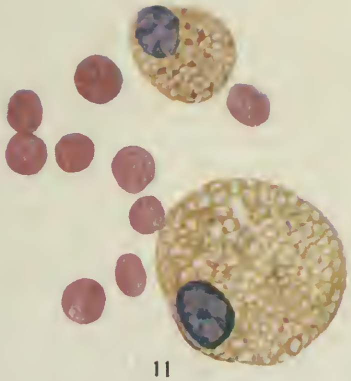

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EXPLANATION OF PLATE 3

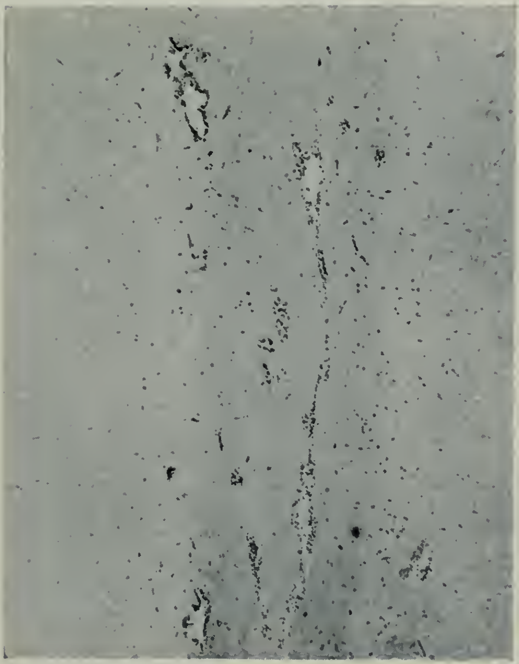
Fig. 12.—Photomicrograph of a longitudinal section of the stab at the six-hour stage — S27-24-6; hematoxylin and eosin stain. The dark strip in the center is the coagulum. Running through it is the cleft made by the needle, and it contains many small blood clots. Its dark appearance is due largely to the dye which it has absorbed. Around this coagulated tissue is the intermediate zone, lighter in tone, and open in texture. Outside of this, again, is the more or less necrotized wall of the brain tissue, which passes gradually into the normal brain beyond; × 60.

Fig. 13.—Photomicrograph from a similar preparation, only at the two-day stage of repair — S27-6-7; hematoxylin and eosin stain. The coagulum, with its blood clot, is seen occupying the center, and around it are the zones already described. The phagocytes, which are invading the field, are not seen in the figure. Two of them are shown in Figure 8, from the edge of the coagulum. The cross in the upper right-hand corner marks the spot from which Figure 24 was drawn. Macrophages are found all through this region. Peculiar extracellular dye granules are found in the coagulum, as shown in Figure 27; × 60.

Fig. 14.—Photomicrograph showing the split stab at the third day of repair— S27-37-6; hematoxylin and eosin stain. The zones are well shown. The most interesting feature is the enormous number of phagocytes in the intermediate zone. Two of these appear in Figure 9. Only the nuclei appear in the illustration. There is some solution of the necrotic tissue. A membrane, isolating the defect from the surrounding brain tissue, will later appear in this region of phagocytic concentration, as the next figure will show; × 60.

Fig. 15.—Photomicrograph showing the split stab at the fifteenth day of repair — S27-18-5; hematoxylin and eosin stain. Note that in this specimen, where the injury was relatively slight, the necrotic tissue has almost entirely been removed, leaving a loose reticulum, in which are a few macrophages. Figures 10 and 11 were drawn from this area. Note the membrane, continuous above with the pia-arachnoid, which cuts off the surviving brain tissue from the defect; × 60.

PLATE 3



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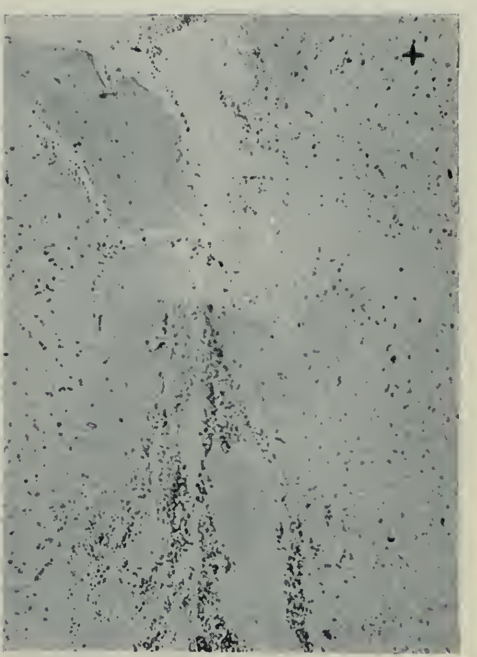

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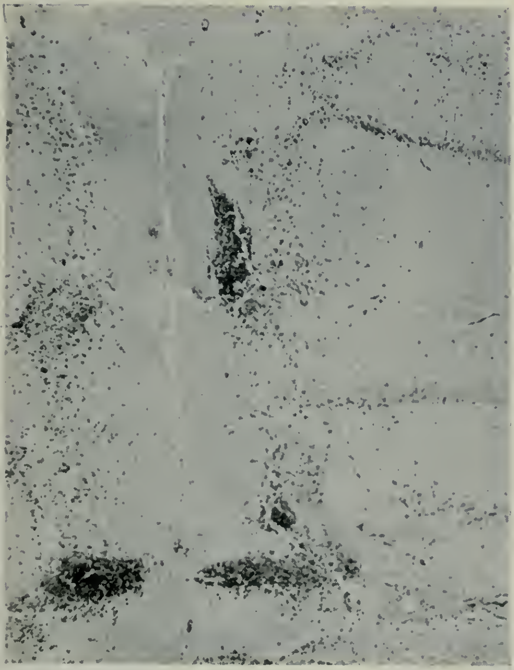

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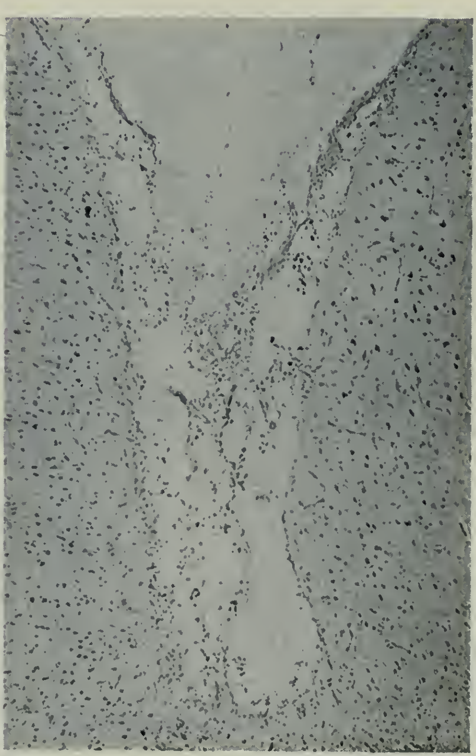

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EXPLANATION OF PLATE 4

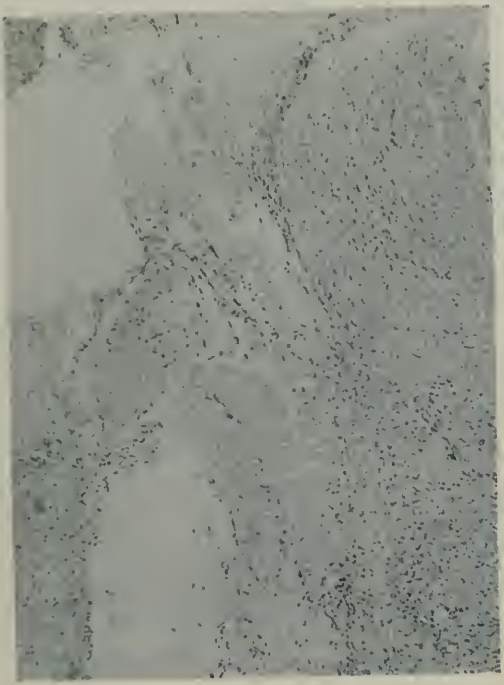
Fig. 16.—Photomicrograph of the split stab at the twenty-fourth day—S27-46-1; hematoxylin and eosin stain. This was a more severe injury than that of Figure 15. The defect is marked by open spaces and the remains of the necrotized tissue, with a reticulum of connective tissue and blood vessels in places. Many macrophages persist, as the next figure shows. Note the membrane which delimits the injured region. It is best marked above; × 60.

Fig. 17.—Photomicrograph of an area from Figure 16, at a higher magnification. Twenty-four-day stage— S27-46-1; hematoxylin and eosin stain. A number of large macrophages are seen. Below is the brain wall, with a short length of the isolating membrane; × 280.

Fig. 18.—Photomicrograph of the defect at the sixty-second day — S27-3-4; hematoxylin and eosin stain. A loose reticulum of connective tissue, and a few shreds of apparently indissoluble coagula, occupy it. The isolating membrane is well marked, and is seen in the next figure more highly magnified. A few macrophages persist; × 60.

Fig. 19.—Photomicrograph of the membrane lining the wall of the defect seen in Figure 18 — S27-3-4; sixty-second day stage; hematoxylin and eosin. The surrounding brain wall is open and reticulated in structure, and the large nerve cells have been destroyed. A few macrophages are seen; × 280.

PLATE 4



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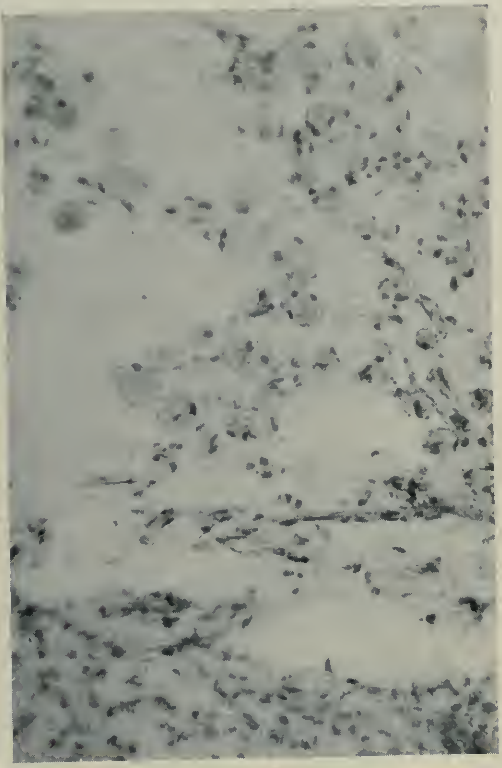

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Explanation of Plate 5

Fig. 20.—Photomicrograph of the stab at the seventy-fourth day of repair—S27-4-4; carmin stain. This injury was made by a slender needle, and was relatively slight. The defect is comparatively trivial. In the thin cleft are a number of pigment containing macrophages, and the limiting membrane is much attenuated. The next figure shows it at a higher magnification; × 60.

Fig. 21.—Photomicrograph of the top of the specimen seen in Figure 20 — S27-4-4; seventy-four-day stage; carmin stain. Note the few macrophages in the defect; × 280.

Fig. 22.—Photomicrograph of small abscess which was found at the twelfth day of repair in one of the S27 cases (S27-41-9); carmin stain. The pus cell accumulation is shown walled in by macrophagic tissue, beyond which is a zone of fibrous tissue, cutting off the brain substance. The area enclosed is seen enlarged in Figure 25. Figure 7 shows this abscess as it appears in the cleared and uncounterstained section, and exhibits the dye content of the macrophagic wall. In Figure 23 a group of macrophages from this wall appears at a higher magnification; × 60.

Fig. 23.—Wash drawing of a group of macrophages from the wall of the abscess shown in Figures 22 and 25 — S27-41; twelve-day stage; carmin stain. Dye granules are seen in jet black in the drawing, but appear blue in the original specimen. Camera lucida; × 780; drawn by M. T. M.

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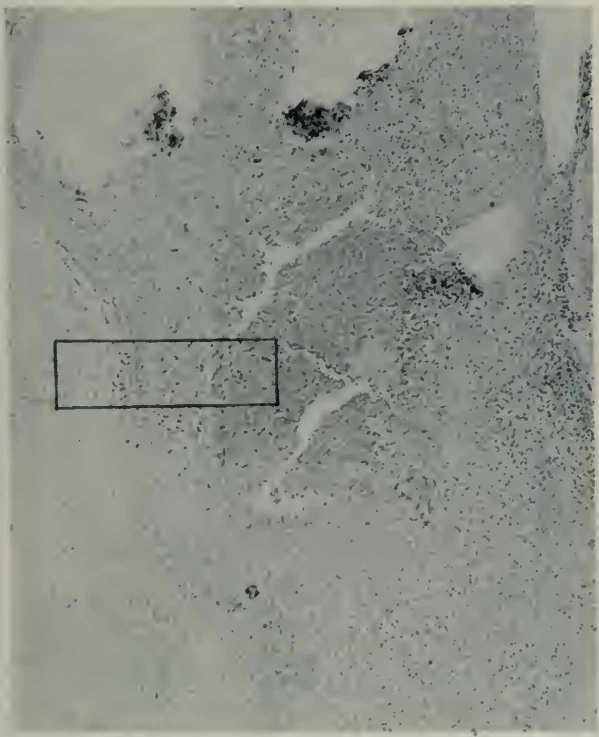

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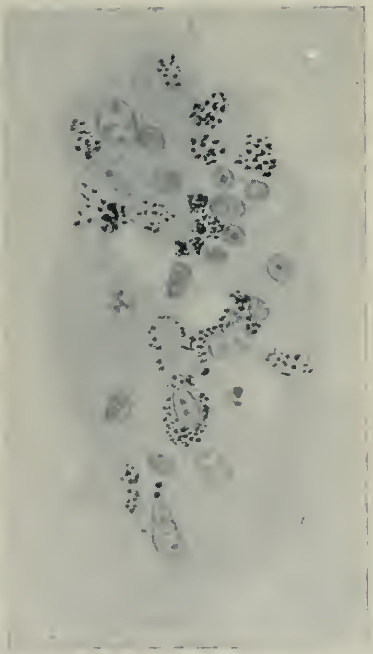

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Explanation of Plate 6

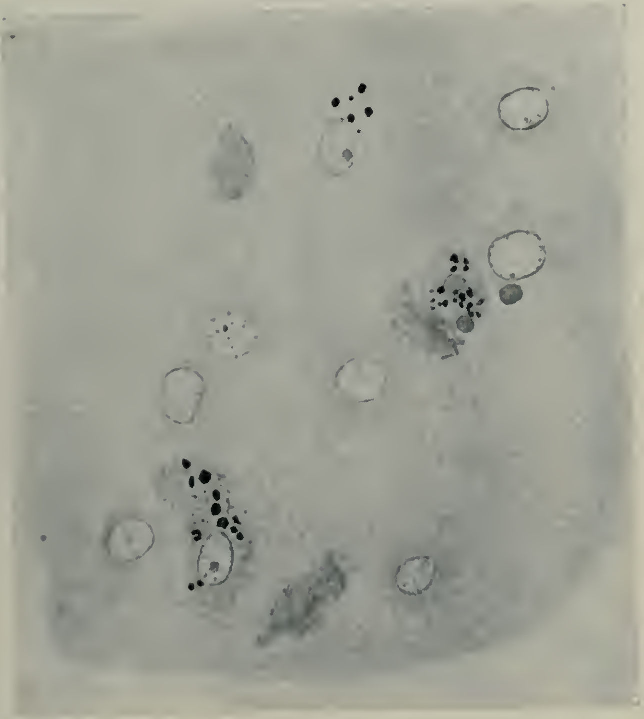
Fig. 24.—Wash drawing of an area of brain tissue from the two-day stage (region shown by cross in Figure 13)— S27-6-6; carmin stain. The tissue has been damaged by the heating, and the ganglion cells are dead. They appear in the drawing as irregular dark masses. A few of the neuroglia cells have taken the vital staining, and contain bright blue granules in the original specimen. These granules are shown in the drawing as jet black. Note the reticulated appearance of the intercellular substance. Camera lucida; × 780; drawn by M. T. M.

Fig. 25. Wash drawing of a panel from the abscess shown as a photomicrograph in Figure 22; S27-41-9; twelve-day stage; carmin stain. From below up we have the pus cell accumulation (*a*); the macrophage wall, with dye granules shown in jet black (*b*); the fibrous membrane (*c*); and the surrounding brain (*d*). Some of the macrophages are shown more highly magnified in Figure 23. Camera lucida; × 340; drawn by M. T. M.

Fig. 26.—Wash drawing of arachnoid, subarachnoid space, pia and underlying brain, from an area near stab, at the second day of repair—S27-6-6; carmin stain. A stab of this specimen is seen in the photomicrograph, Figure 13. Two pia cells are shown somewhat enlarged, and contain dye, which is depicted as jet black granules. Camera lucida; × 780; drawn by M. T. M.

Fig. 27. — Crayon drawing of an area of coagulum in which small dye granules of varying size appear (shown in jet black). Section cleared without counterstaining — S27-6; two-day stage. In the original the background is of pale diffuse blue, while the granules are strongly blue. The intensity of staining of the granule varies. Some of the granules are in small clumps. A few red blood cells are seen. A photomicrograph of this stab appears in Figure 13. Camera lucida; × 1500; drawn by C. C. M.

Fig. 28.—Wash drawing of a frond of the choroid plexus on the injured side at the second day of repair — S27-6-6; carmin stain. The same specimen is shown in the photomicrograph in Figure 13. The macrophages are found densely stained with dye granules; three are seen in the figure. A few stained granules in the epithelium appear also. All dye granules are shown in jet black. Camera lucida; × 780; drawn by M. T. M.



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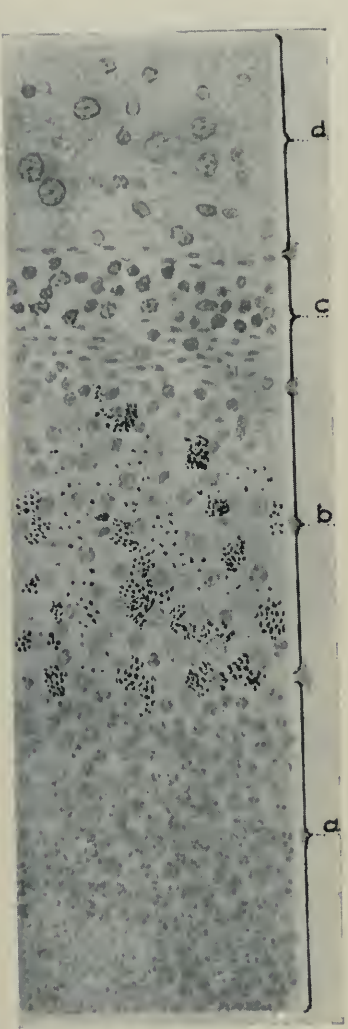

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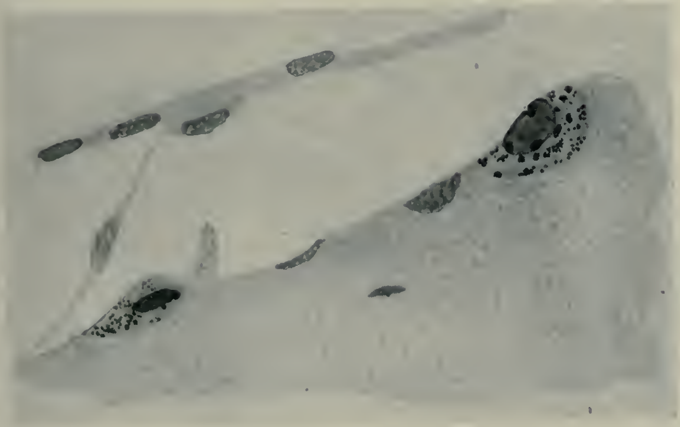

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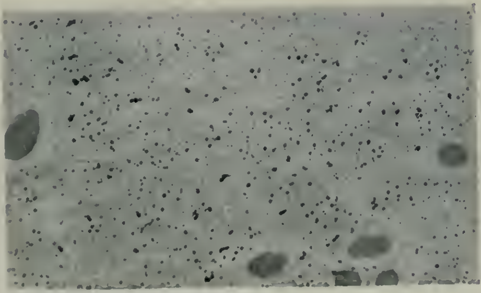

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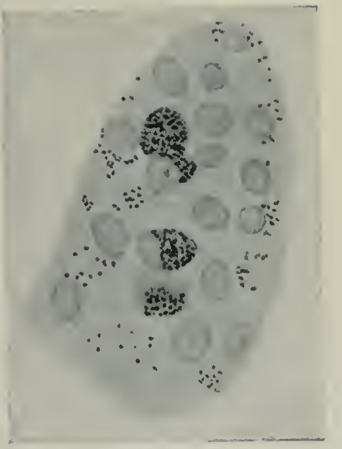

28

A CASE PRESENTING AN EPIDERMOID PAPILLARY CYSTOMA INVOLVING THE THIRD VENTRICLE *

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GENERAL NEUROPATHOLOGY OF THE CASE

Epidermoid growths involving the third ventricle have created particular interest with reference to their relation to the pituitary body and the disturbance of its function, the possibility of localization of such tumors, and their source of origin.

Recalling the epiblastic origin of the neural tube, one is not surprised to find in close relation to the central nervous system, ectodermic cells originally intended for skin and mucous membrane. They may develop as if they were in their normal location with the formation of dermoid cysts or cholesteatomas.

The presence of epidermoid tissue in the third ventricle may be explained as being the result of an infundibular anlage. Cushing[1] reports two examples of infundibular cysts filled with a yellowish gelatinous substance, and with numerous verrucose nodules composed of squamous epithelium projecting from the walls, which he considers as possible developmental aberrations in relation to the neurohypophysis. Similar infundibular cysts were described by Langer[2] and Strada.[3]

Saxer[4] reported a tumor which he believed originated from the epithelial lining of the ventricle, or the pars intermedia of the pituitary body, the gland being normal.

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* From the Massachusetts State Psychiatric Institute, Massachusetts Commission on Mental Diseases. This paper was one of a series presented to Dr. E. E. Southard on the occasion of the tenth anniversary of the establishment of the Bullard Professorship of Neuropathology, Harvard Medical School, 1906 to 1916.

[1] Cushing, H.: The Pituitary Body and Its Disorders, Philadelphia, J. B. Lippincott Company, 1912, p. 289.

[2] Langer, F.: Ueber Cystische Tumoren im Bereiche des Infundibulum Cerebri, Ztschr. f. Heilk. **13**:57, 83, 1892.

[3] Strada, F.: Beiträge zur Kentniss der Geschwülste der Hypophyse und der Hypophysengegend, Virchows Arch. f. path. Anat. **203**:1, 1911.

[4] Saxer, F.: Zeiglers Beiträge **32**:333, 1902.

Mott[5] mentions the possibility of epidermoid tissue being transported by the vessels which grew into the third ventricle to form the choroid plexus, also of its having arisen from cells of the hypophyseal diverticulum.

The hypophysis or anterior lobe of the pituitary body originates as a pharyngeal diverticulum (Fig. 1). The exact location of its origin with reference to the juncture of the pharyngeal and buccal epithelium is still a matter of dispute. Minot[6] considered the diverticulum an outgrowth from the ectodermal lining of the mouth. Normally, the cells forming the original stalk atrophy and the hypophysis loses all connection with the epithelium of the oral cavity. It is not uncommon, however, to have a retention of some cells along the stalk tract, and Erdheim[7] in cross sections of normal glands found inclusions of pavement or ciliated cells near the superior and inferior portions of the hypophyseal cleft and within the gland, presumably representing "rests" of the primitive ectodermic diverticulum. He recorded seven heteroplastic tumors probably arising from such "rests."

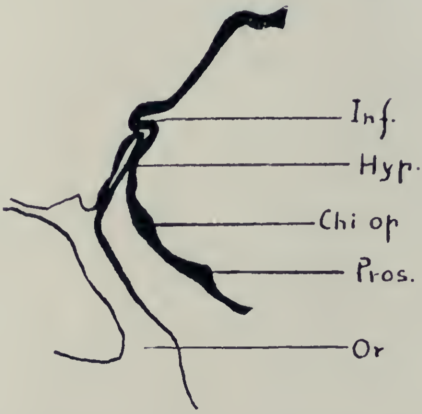

Fig. 1.—Origin of the hypophysis as a pharyngeal diverticulum: Inf., infundibulum; Hyp., hypophysis; Chi. op., optic chiasm; Pros., prosencephalon; Or., mouth.

Dean Lewis[8] emphasizes the frequency and importance of tumors arising from craniopharyngeal duct epithelium; reviews the literature with special attention to cases reported by Wagner, Langer and Harbitz, and describes a case. He presents a table by Creutzfeldt, published in 1908, showing that in fifty-five necropsies of tumors of the hypophysis without acromegaly, nineteen were classed as craniopharyngeal duct tumors.

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[5] Mott, F. W., and Barrat, J. O. W.: Arch. of Neurol **1**:417, 1900.

[6] Minot, C. S.: A Laboratory Textbook of Embryology, Philadelphia, P. Blakiston's Son & Co., 1910, p. 292.

[7] Erdheim, J.: Ueber Hypophysengangsgeschwülste und Hirncholesteatome, Sitzungsb. d. k. Akad. d. Wissenschrift Math.-naturu. **150**: 1904; **113**:537.

[8] Lewis, D. D.: A Contribution to the Subject of Tumors of the Hypophysis, J. A. M. A. **55**:1002 (Sept. 17) 1910.

In the thirty cases of tumor of the third ventricle reported by Weisenburg,[9] only three are stated to be epidermoid in character. These include the cases of Saxer and Mott.

In the case here presented, the growth did not show the large polygonal cells with densely staining protoplasm, characteristic of tumors of the pars intermedia, nor did it resemble those arising from the choroid plexus. The infundibulum was distended, and its tissue partially replaced by the tumor with a suggestion of a tumor stalk near the right ventrolateral surface. Cross sections through the third ventricle and pituitary body, which was removed intact still attached to the brain, showed the dura-like capsule of the growth to be continuous with the connective tissue of the gland. The pituitary gland was normal. The squamous epithelium of the tumor showed a suggestion of intercellular spines and well marked scaling; but no hair or sebaceous material was found. Vacuolization and cilia formation are not differential, and there does not seem to be any adequate criterion by which one may judge how this epidermal tissue happened to be in this location. From the situation and character of the growth, it probably originated either as a result of a developmental abnormality of the infundibulum or from an hypophyseal "rest."

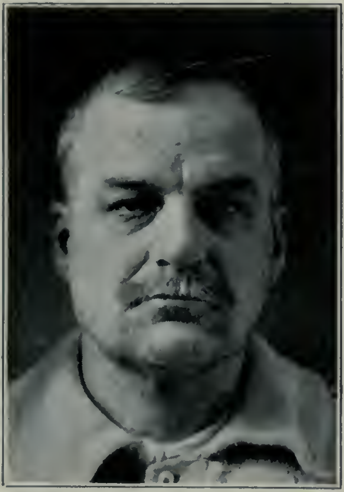

Fig. 2.—Patient, May 10, 1914.

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[9] Weisenburg, T. H.: Tumors of the Third Ventricle with the Establishment of a Symptom Complex, Brain **33**:236, 1910.

In the grouping of third ventricle tumors according to symptomatology, suggested by Weisenburg[9] in an excellent review of thirty cases of which he reports three, this case would be in Class 1, i.e., those cases in which a tumor of moderate size is situated in the floor of the third ventricle and in which there is no extension into the foramen of Monro or the aqueduct of Sylvius. Though the aqueduct was not dilated, the posterior part of the ventricle and the peri-aqueductal structures were apparently involved indirectly as shown by the pupillary disturbance without, however, paralysis of associated ocular movements and a reeling gait suggestive of involvement of the red nuclei or superior cerebellar peduncles.

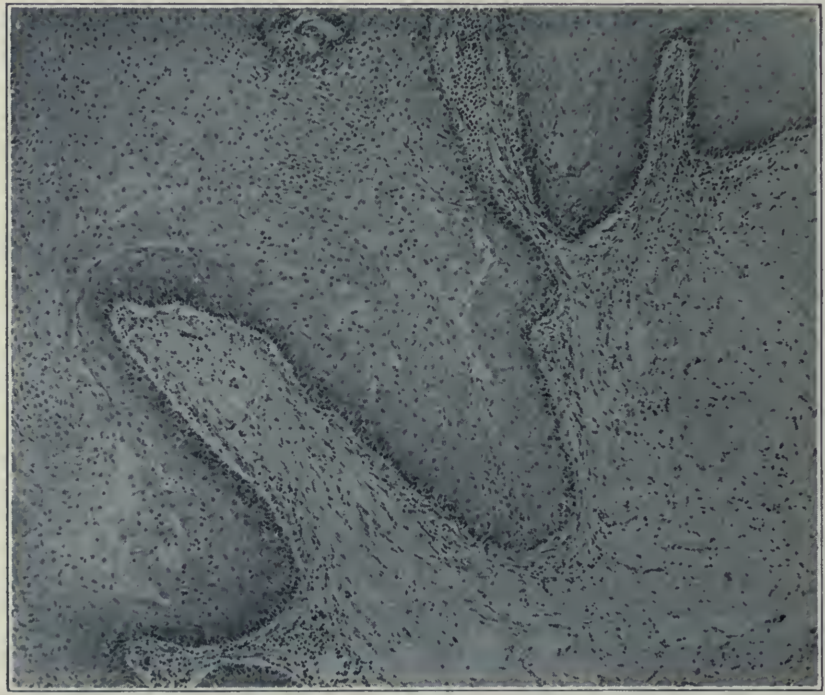

Fig. 3.—Papillomatous character of the growth, type of epithelium and connective tissue stroma. Hematoxylin and eosin, × 70.

The patient's tendency to drag his feet and the weakness of the legs might be interpreted either as evidence of pressure on the internal capsules or of cortical injury.

One of the early symptoms in this case was the evidence of hypopituitarism (loss of sexual power, transient polydipsia and polyuria, and possible gain in weight). These may have been due to the fact that, occupying the base of the third ventricle, the growth at an early date interfered with the discharge of a secretion of the posterior lobe of the pituitary gland directly into the cerebrospinal fluid, or to disturbance of function due to pressure on the whole gland. Histologically, no change was noted.

Another striking feature was the drowsy, somnolent, apathetic condition of over a year's duration, with periods approaching normality. Weisenburg does not consider this feature as specific of lesions of the third ventricle, but due to impairment of the normal function of the cortex, the result of pressure against the skull through dilatation of the lateral ventricles. Mott also believed it to be the result of compression of the cortex by internal hydrocephalus, and explained the recurrences by the escape of fluid from the lateral ventricles, thereby relieving the cerebral compression and removing the cerebral anemia. Purves Stewart[10] considered that somnolence in these cases was associated either with direct upward pressure on the third ventricle or with secondary anemia from compression of the vessels at the base of the brain.

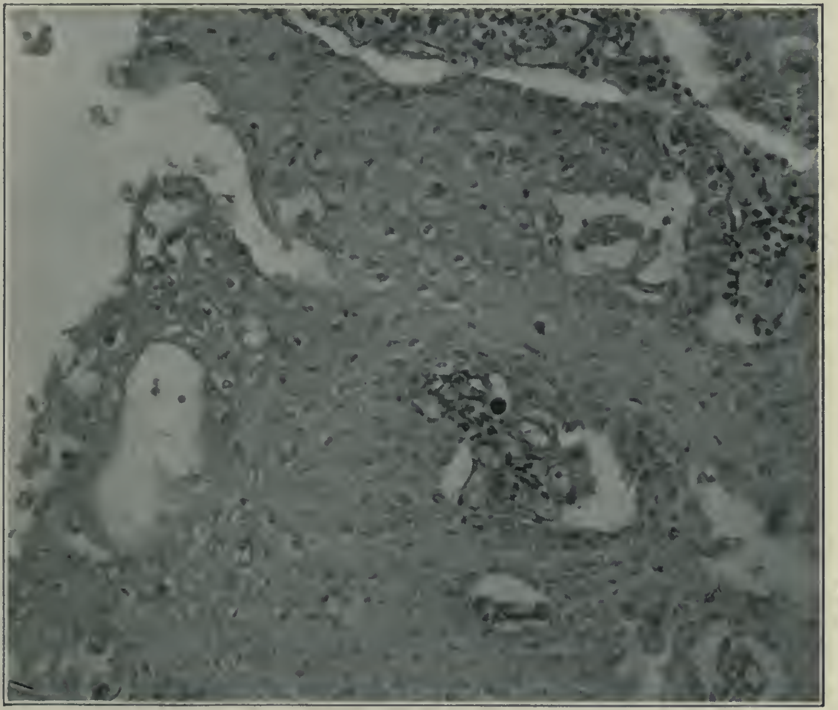

Fig. 4.—Inner aspect of cyst cavity. Hematoxylin and eosin, × 215.

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[10] Stewart, Purves: Four Cases of Tumor in the Region of the Hypophysis Cerebri, Rev. Neurol. & Psychiat., April, 1900, p. 239.

Turner[11] thought that it was due to cerebral edema. Cushing[12] notes hypersomnia in many cases of hypopituitarism and suggests, though certain cases showed sufficient increase in cerebral tension to possibly account for the drowsiness, that the condition may be associated with changes in glandular function. This relation has been considered as an explanation of normal sleep. The case reported by Purves Stewart in which a sarcoma (extracerebral) destroyed the pituitary gland without clinical evidence of somnolence, is in opposition to this theory.

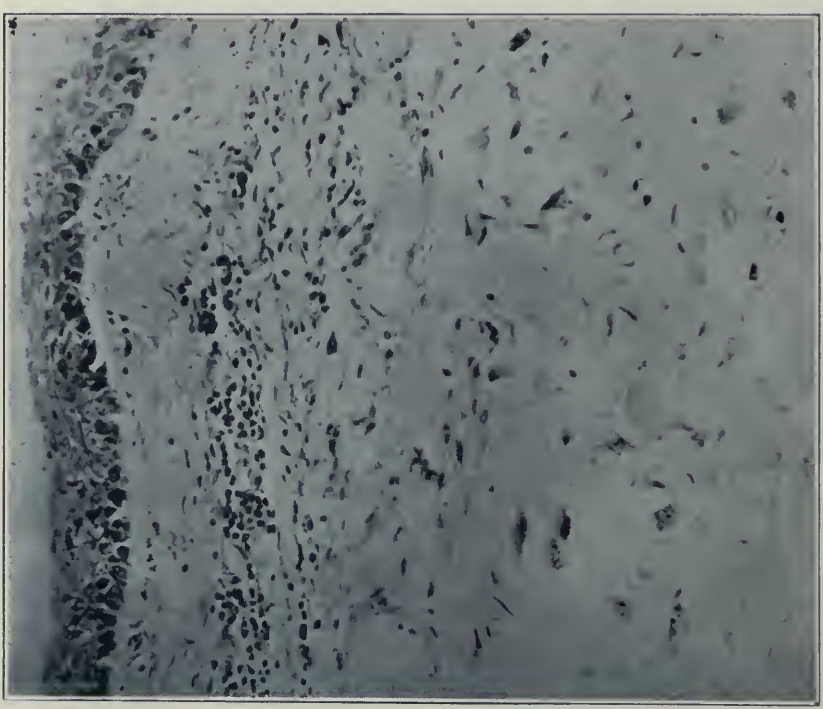

Fig. 5.—Relation of the tumor to the thalamus. Cellular infiltration of the connective tissue. Hematoxylin and eosin, × 225.

In the case under consideration there was an internal hydrocephalus with increased intracranial pressure, cerebral anemia, edema and pituitary disturbance: it was not a suitable case for determining whether somnolence is a general diffuse effect, or results from the involvement of a focal center, possibly in the floor of the third ventricle or pituitary body.

The mental condition (change in disposition, enfeeblement of memory, disorientation, untidiness, etc.) is compatible with the fact that the chief gross evidences of pressure were in the prefrontal areas. Sections from the second and third frontal gyri on the left showed loss of myelin in the fibers of the zonal layer. There was a slight diminution in the number of cortical cells, and those present showed considerable chromatolysis.

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[11] Turner, J.: Rev. Neurol. & Psychiat., June, 1910, p. 351.

[12] Cushing: (Footnote 1) p. 269.

Areas Involved by Tumor.—The tumor directly involved by pressure the gray substance and fiber tracts about the third ventricle including:

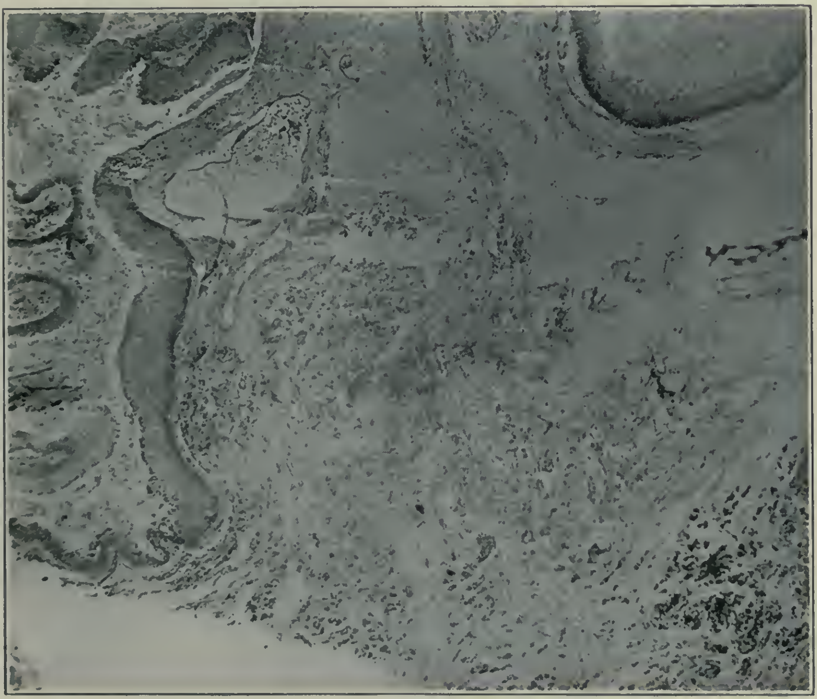

Fig. 6.—Relation of base of growth to the pituitary gland. Globule of hyalin material. Hematoxylin and eosin, × 23.

(*a*) The anterior commissure containing fibers between the two hippocampal regions (pars temporalis), and those derived from the lobus olfactorius connecting the olfactory tract on the one side with the hippocampal region on the opposite side (pars olfactoria).

(*b*) The fornix composed of fibers arising in the hippocampal region, pursuing an arched course to the corpora mammillaria. Some fibers terminate here, others cross the midline and turn downward into the reticular formation as far as the pons.

(*c*) The lamina cinerea and lamina terminalis, a thin layer of gray substance between the corpus callosum and chiasma, continuous above the chiasm with the tuber cinereum and connected at the side with the gray substance of the anterior perforated space.

(d) The optic chiasm and the optic tract, especially on the left, were distorted and flattened. The disks showed a marked papilledema and deposition of new tissue.

(*e*) The tuber cinereum — a lamina of gray substance extending forward from the corpora mammillaria to the optic commissure to which it is attached. It presents several small collections of ganglionic

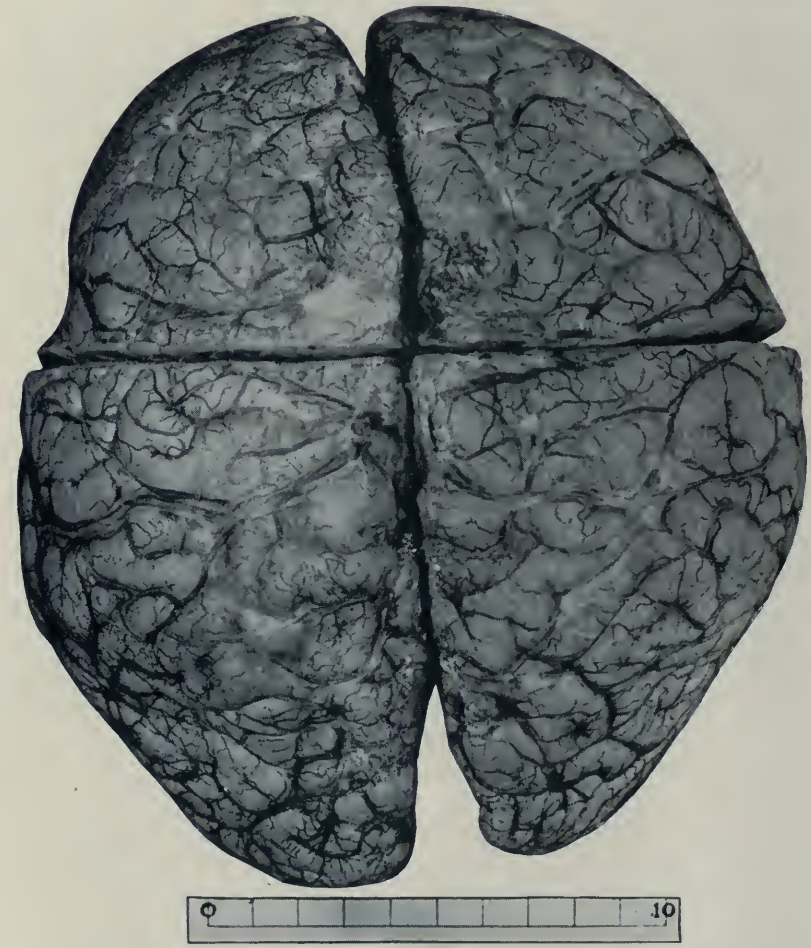

Plate A.—Upper surface of the brain. The brain before fixation tended to assume a round form when placed on a flat surface. The brain surface was dry, with a tracing of irregularly injected vessels. The convolutions were pressed out in appearance, the sulci being indicated on the right. The swelling of the brain and the widening and flattening of the gyri were most marked anterior to the posterior parietal lobules. The mesial surface also showed the flattening of the convolutions, especially on the left in the prefrontal region.

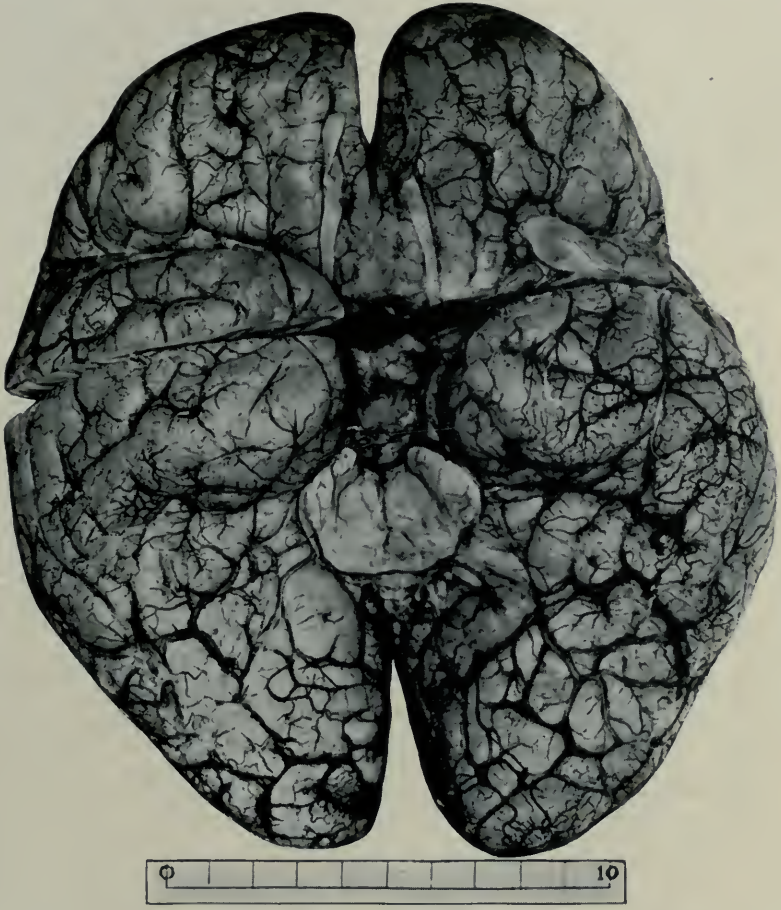

Plate B.—Lower surface of the brain. The vessels were small with inequality of the vertebrals. The left middle cerebral appeared small, and its lumen tiny. There was some hyalin change in the middle cerebral vessels, but no sclerosis. The olfactory bulbs were red and slightly unequal. The optic chiasm measured 2 by 1 cm.; it was much distorted as though from pressure beneath. The optic tract on the left was broad and bulging, the right less so. The optic nerves were flattened. The tuber cinereum was translucent gray. There was a small nodular protrusion of tissue on the right. The pituitary was normal in size and consistence. There were several grayish-yellow spots on the surface. The mammillary bodies were slightly flattened.

The pituitary gland, removed attached to the brain, appeared normal in size and outline.

The mammillary body on the left appeared swollen as though from pressure from beneath.

The third nerves were involved in a thickening of the pia between the peduncles. The brain stem in sections at the level of the red nuclei which appear as circular areas just dorsal to the substantia nigra and crusta.

The cerebellum appeared normal.

cells; the lateral eminences; the eminentia vascularis, homologue of the saccus vasculosus of lower vertebrates, especially prominent in the fishes. Johnston[13] considers the eminentia vascularis as possibly an organ for controlling the character of the spinal fluid. The basal optic ganglion (supraoptic nucleus Cajal)—a tract of gray matter with nerve cells, lying to the outer side of the tuber cinereum close to the optic tract. From each a tract issues, which, after decussating with that of the opposite side (Meynert's commissure), applies itself to the mesial side of the optic tract and passes back to the lenticular nucleus. (Déjerine.)

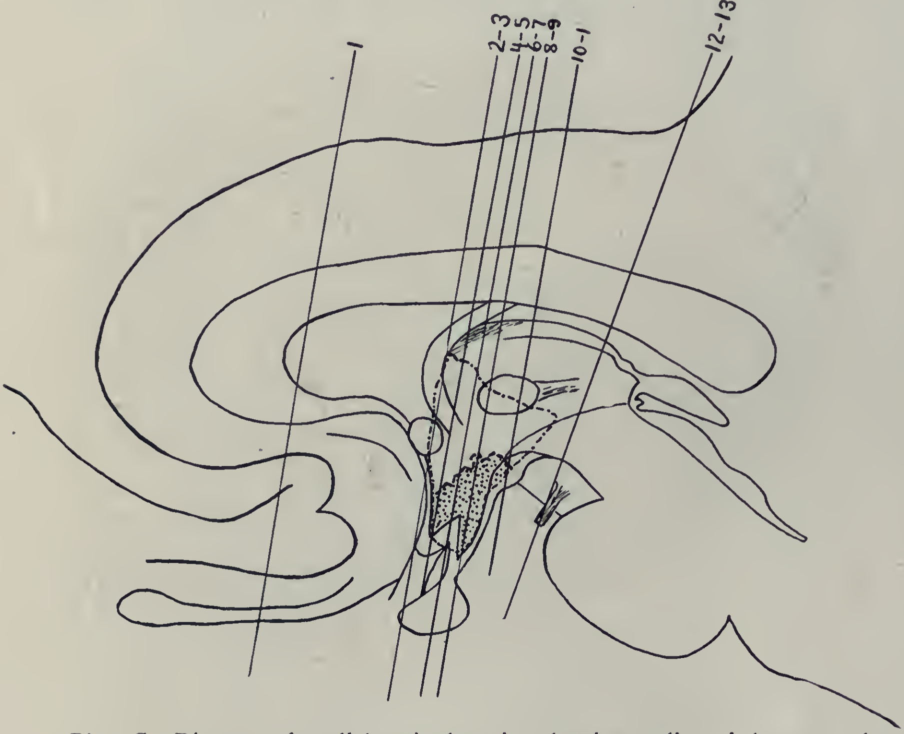

Plate C.—Diagram of medial sagittal section showing outline of the cyst and levels at which the following frontal sections were made.

In plates 3, 5, 7, 9, 11 and 13 one is looking toward the posterior pole of the brain.

In plates 2, 4, 6, 8, 10 and 12 one is looking toward the anterior pole of the brain.

(*f*) The corpora mammillaria, especially the mesial nuclei. Efferent tracts: Bundle of Vicq d'Azyr to the dorsal or anterior nucleus of the thalamus; bundle of Gudden to the red nucleus and adjacent gray matter of the tegmentum.

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[13] Johnston, J. B.: The Nervous System of Vertebrates, Philadelphia, P. Blakiston's Son & Co., 1906, p. 285.

Afferent tracts:[14] (*a*) by way of the columns of the fornix, and (*b*) through its peduncle from the main fillet and arcuate fibers in the tegmentum.

(*g*) Posterior thalamic decussation,[15] behind the mammillary bodies, probably a connection between the corpora Luysii. (Déjerine.)

(*h*) The intermediate gray mass or middle commissure, uniting the mesial nuclei of the thalamus across the third ventricle and continuous below on each side with the gray matter of the cavity.

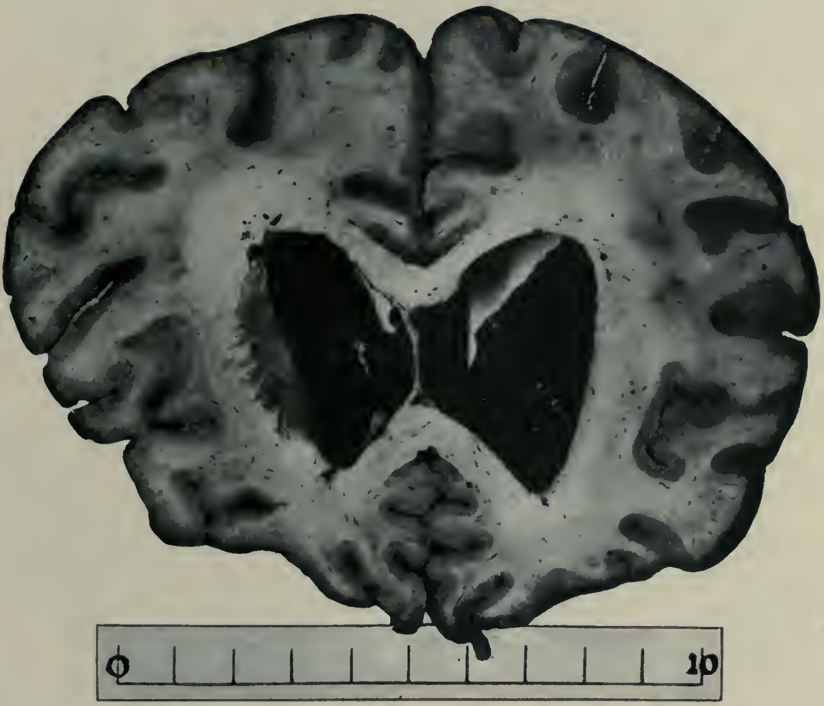

Plate 1.—Frontal section just posterior to the genu corporis callosis. Slightly oblique. The septum pellucidum extends between the truncus and rostrum corporis callosi. Lateral to the anterior horns of the ventricles is the stratum subependymale and on the right the corpus striatum before its division into the nucleus caudatus and the nucleus lenticularis by the traversing fiber bundles of the internal capsule. Converging toward the corpus striatum are seen the strands of the corona radiata. The olfactory bulb and tract are folded back beneath the section. The increased size of the left ventricle may be noted.

(*i*) The mesial series of nuclei of the thalamus (Cajal). There was no suggestion of a thalamic syndrome in this case.

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[14] Quain, Jones, and Quain, Richard: Elements of Anatomy, Ed. 11, New York, Longmans, Green & Co. **3**:232, 1909, Part 1.

[15] (Footnote 14) p. 228.

(*j*) Hypothalamus[16]— the prolongation of the tegmentum under the posterior part of the thalamus, divided by Forel into three layers from above down — the stratum dorsale, zona incerta and the corpus subthalamicum or nucleus of Luys, the latter having taken the place of the substantia nigra lying next to the prolongation of the crusta.

(*k*) The internal capsule. The possibility of determining capsular involvement was complicated by the cortical injury.

It has been difficult to estimate the degree of injury suffered by the tissues adjoining the growth, and even more difficult to correlate the injury with any clinical manifestations because of the diffuse area involved and the paucity of data as to the normal physiologic function of the structures.

CLINICAL DETAILS

The case was under the direction of Dr. Frankwood E. Williams.

History.—The history of the patient, a man, aged 52, white, was obtained from his wife. The family history was not important. He had been subject to headaches and vomiting in early life. At 33 he had typhoid fever. His application for life insurance had been rejected six years before because of albuminuria. He had pertussis in 1913. He denied venereal disease. He had lost sexual power one and one-half years before. He used no alcohol, and no drugs, but smoked a cigar occasionally.

The patient had been well until August, 1913, when he had occasional severe frontal headaches and complained of not feeling well. During the following months he had frequent headaches accompanied by constant nausea, but not by vomiting; he was abnormally sleepy and extremely irritable. In February, 1914, he drank large quantities of water, sometimes eighteen glasses in an hour; he again became lethargic and confused mentally, but was not considered seriously ill. He gave up his work. In April, he was frequently disoriented for time, his memory was poor, and his vision seemed to be failing. His speech became thick; he began to drag his feet, and he developed incontinence of urine.

He was admitted to the Psychopathic Department of Boston State Hospital, May 7, 1914. At first, he was excited, tore his blankets and sheets into strips and tied them in a fantastic manner about his feet. He was oriented for person, but not for place or time. There was a marked loss of memory for both recent and remote events with a tendency to fill the gaps with fabrications. He had some delusions of grandeur, magnifying the amount of his wealth and assuring the staff that he built all the buildings around the hospital. Definite evidence of hallucinations was not obtained. Emotionally, he ordinarily had been self-reliant, very cheerful, even-tempered, social (got on well with people), reserved but never seclusive. At the time of examination he appeared self-satisfied, somewhat euphoric and inclined to be restless. He had no insight into his condition.

Examination.—Physical: The patient was a well developed and well nourished man, 5 feet 8 inches in height, weighing 203 pounds, stripped. His face was flushed. The general examination was negative except for very slight edema over the feet and shins. The systolic blood pressure was 150 mm. of mercury.

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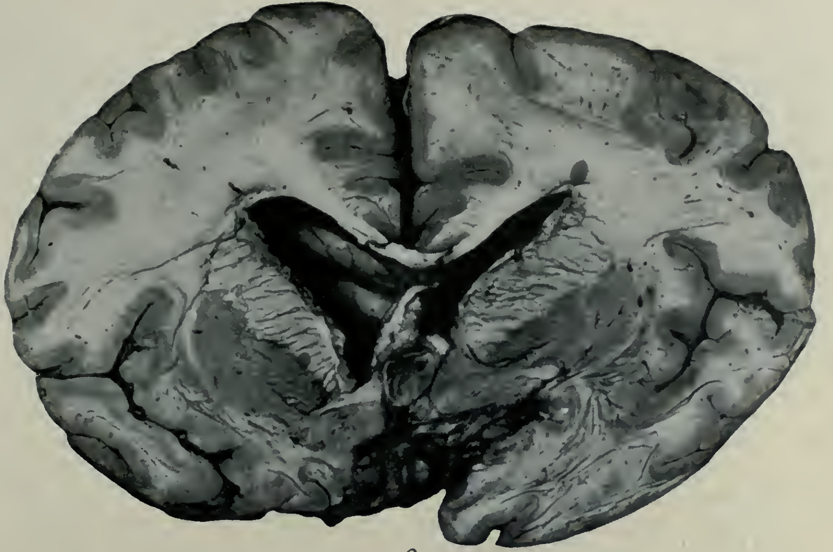
[16] (Footnote 14) p. 250.



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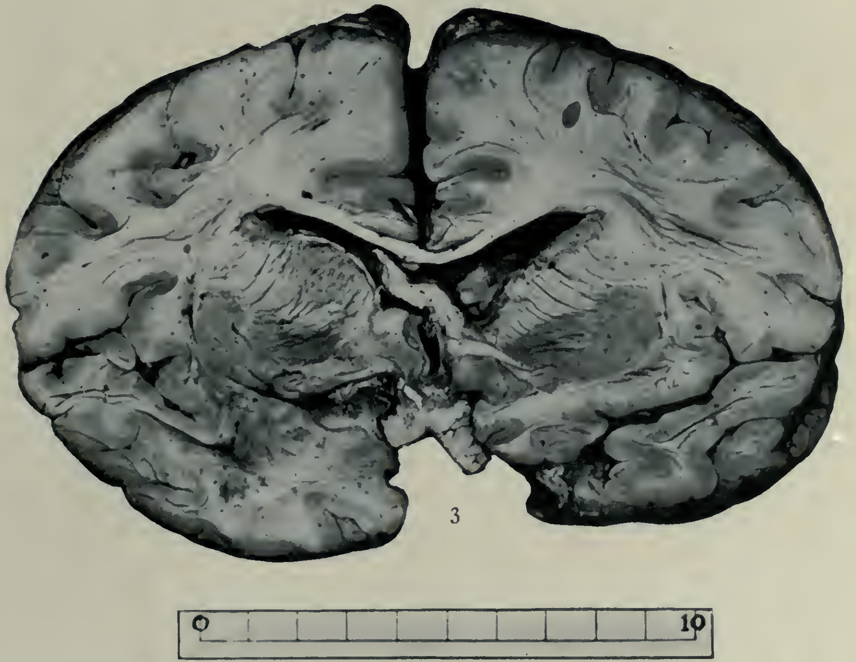

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Plates 2 and 3.—Oblique section. Left side shows more anterior level than the right. The internal capsule appears dividing the corpus striatum into the now small caudate, and the lenticular nuclei. The latter shows a subdivision into the globus pallidus and the putamen. On the right ventral to the corpus striatum lies the substantia perforata anterior, on the left the tuberculum olfactorium. The fibers of the anterior commissure are seen crossing the ventral part of the globus pallidus on the left, more laterally on the right. Ventral to the septum pellucidum appear the fiber bundles of the fornix, more prominent on the left.

Posterior to the recessus triangularis, rather loosely held to, but distorting, the columnae fornicis, anterior commissure and lamina terminalis is seen the sacular termination of the cyst occupying the base of the third ventricle. The foramen of Monroe is seen on the left. The increased size of both lateral ventricles may be noted.

Neurologic: The pupils were central, the right slightly larger than the left, and both were irregular. Consensual reaction and reaction to direct light were sluggish. Accommodation was normal. Extra-ocular movements were normal. There was no ptosis, strabismus or nystagmus. Examination was made of the fundi by Dr. Gerhardt in consultation, May 25, 1914. There was slight hyperemia of the disks, not pathologic. The deep reflexes were normal. Abdominal and cremasteric reflexes were not obtained. The gait and station were normal. There were no areas of anesthesia or hyperesthesia. There was no tenderness over the nerve trunks. Muscle sense was retained except for slight inaccuracy in the finger to nose test.

Laboratory Studies.—Urine: An occasional slight trace of albumin and a rare hyalin cast were found in the urine. A study of the acidity by the hydrogen-ion concentration method[17] gave evidence of a slight acidosis (plus 0.9, two hours after 4 gm. of bicarbonate of soda). The phthalein output was 18 per cent. The time was not stated.

Blood: The Wassermann reaction was negative. The cytologic examination was negative. Quantitative examination of uric acid, urea and nonprotein nitrogen showed a nitrogen retention (100 per cent. increase over the maximum normal values as given by Folin and Denis[18]).

Spinal Fluid: Three examinations were made. Cells, 2 to 6. Globulin was negative, and albumin was found in small amounts. The Wassermann reaction was negative. The colloidal gold test was negative for syphilis. There was 100 per cent. total nonprotein nitrogen increase. Urea at the pathologic threshold, based on normal values given by Mestrezat.[19]

Head: Roentgenographic examination of the head was negative, June 24, 1914.

Course of the Disease.—During the spring and summer, the patient remained confused, retarded and apathetic, took no care of his person, was untidy, and was frequently found lying about exposed. In October, because of the slight acidosis shown by the laboratory studies, he was given sodium bicarbonate by mouth. He became oriented for time, place and person; his memory improved, and he appeared to take more interest in current events. There were still periods when he was confused and when he had some difficulty in locomotion. At times he would walk like a drunken man. He had lost 39 pounds. Though not well, he was allowed to go home. In three weeks, he returned in a confused state, continually incontinent and very untidy. He remained in bed and refused to eat unless fed. There were a few short periods when he would brighten up and seem to know what was going on about him.

Jan. 3, 1915, after a febrile period of three days (white count 16,800), he became stuporous and died.

Summary.—The patient was a man of 52 years, who had been ill for sixteen months, and whose past history was negative except for typhoid fever nineteen years before and pertussis one year before the present history. There were loss of sexual power; rather acute onset of severe frontal headaches, with nausea but no vomiting; irritability and drowsiness, with increasing mental confusion; transient polydipsia; speech defect; difficult equilibration; distur-

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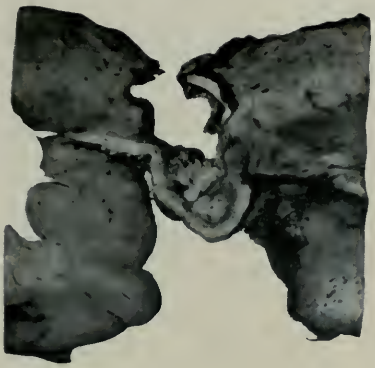
[17] Palmer, W. W., and Henderson, L. J.: Clinical Studies on the Acid Base Equilibrium and the Nature of Acidosis, Arch. Int. Med. **12**:162 (Aug.) 1913.

[18] Folin, O., and Denis, W.: Protein Metabolism from the Standpoint of Blood and Tissue Analysis, J. Biol. Chem. **14**:29 (Feb.) 1913.

[19] Mestrezat, W.: Le Liquide Cephalo-Rachidien, Paris, 1912, pp. 108, 249.



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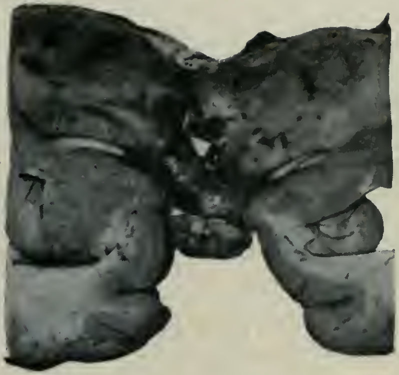

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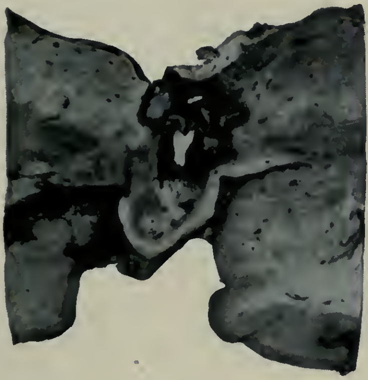

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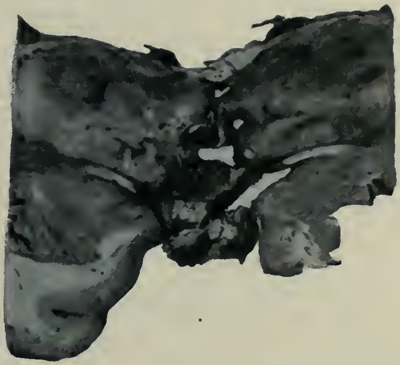

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Plates 4 and 5.—On the left the section strikes through the anterior end of the thalamus. The columnae fornicis lie lower on both sides while the crurae assume a dorsal position. Their attachment to the corpus callosum has been torn. On the right, the thalamus exhibits differentiation into the nucleus anterior, and the nucleus lateralis and the nucleus amygdalae appear within the temporal lobe. The third ventricle is increased in size, irregular in outline and almost completely replaced by the cyst. There is a free upper surface which lies folded on itself across the distended upper portion of the ventricle. The grayish limiting wall can be traced in apposition with the remaining structures about the ventricle. The basal portion of the cyst contains a whitish cauliflower-like growth displaçing the fibers of the optic chiasm, more markedly on the left, and causing the rounded swelling of the left optic tract noted in the description of the base of the brain. Around that portion of the growth, extending into the pocket in the optic tract, is a semicircular mass of colloid material.

Plates 8 and 9.—Section passes through the anterior end of the massa intermedia which is flattened by the distention of the third ventricle and pressure of the cyst which protrudes into it from below involving chiefly the mesial nucleus of the thalamus and bundle of Vicq d'Azyr on the right. There is a small pocket between the cyst wall and the overlying commissure on the left. The portion of the third ventricle above the massa intermedia is flattened but patent. The infundibulum is filled with the growth which bulges into the left side. The pituitary in section shows grayish areas. Microscopic preparations were made from this section.

bance of vision; disorientation; memory defect with tendency to fabrication; expansive delusions; restlessness; untidiness, with a lack of appreciation and indifference to his condition.

Physical Signs: The right pupil was larger than the left. Both were irregular and reacted sluggishly to light. There was a slight speech defect. There were tremor of the extended fingers, increasing weakness of the legs, a tendency to drag the feet, and a loss of control of both the rectal and vesical sphincters.

Laboratory Findings: All tests for syphilis were negative. A slight trace of albumin and casts was found in the urine. There was low kidney function. The blood pressure was 150 systolic. There was retention of nitrogenous products in the blood and spinal fluid, and an increased white count shortly before death.

The case was presented at a meeting of the staff of the Psychopathic Department, May 16, 1914. It created considerable interest and discussion; but no consensus of opinion was reached. It was noted that the case clinically resembled general paralysis; but such diagnosis would hardly be ventured with all tests for syphilis negative. The picture in some respects simulated the terminal stage of Korsakoff's syndrome, but presented no etiologic factor, as alcohol or other toxic agent. No definite indication or localizing sign of arteriosclerosis was present. The evidence of chronic infection, pyogenic or tubercular, was not convincing. Brain tumor was considered; but there were negative eyegrounds, and no localizing signs; an intracranial growth was considered improbable. Although the findings were not typical of uremia, because of the low kidney function, retention of nitrogenous products in the blood and spinal fluid and urinary findings, the case was classified as a cardiorenal psychosis.

Necropsy.—Two hours after death, necropsy was performed by Drs. Solomon, Bunker and Bloomer.

A summary of the positive findings with reference to the body is noted in the anatomic diagnosis.

Kidneys: They weighed 240 gm. They measured 11 by 5 by 2.5 cm. Five small cysts dotted the surface. The capsules were slightly adherent, and the pyramids were poorly differentiated.

Thyroid: It was firm, and section showed nothing of note.

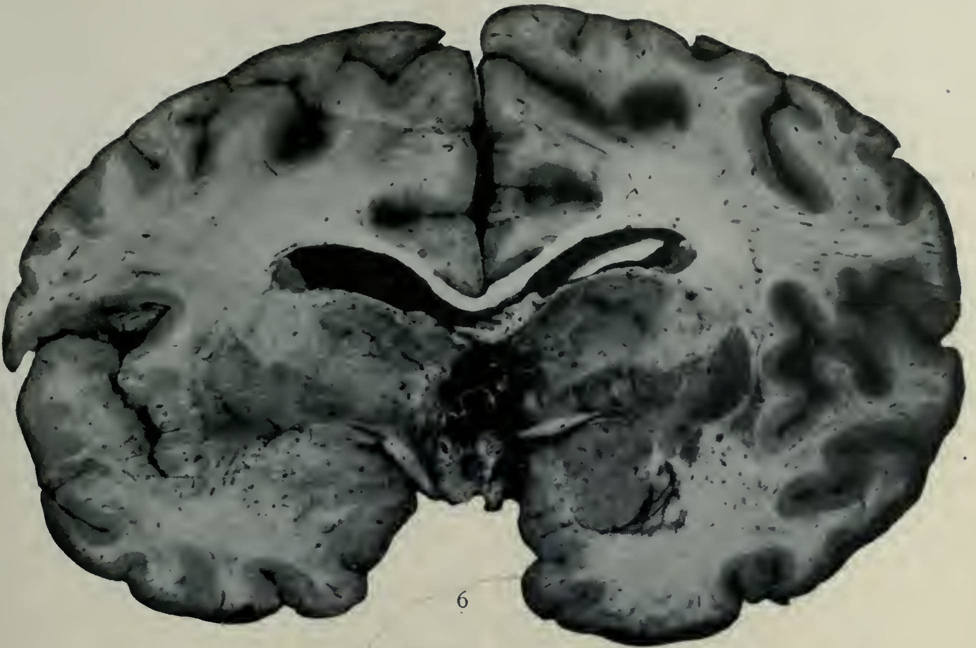
Suprarenals: They were very small, the cortex measured 0.1 by 0.2 cm. in thickness. Two lines of the cortex approximated in one limb; the second limb was separated by a brownish medulla which showed a slight hyalin change in one suprarenal.

Pancreas: It was normal.

Testes: Threaded well.

Head: Description of the brain is given by Dr. Canavan. The hair was somewhat scant. The periosteum was negative. Calvarium measurements were: frontal, 1 cm.; temporal, 0.4 cm., and occipital 0.7 cm. Depressions for the pacchionian bodies were slightly to the right of the median line and pierced the inner table at the vertex. Grooving for the middle meningeals was shallow on both sides.

The dura was not adherent, but was tense; and the brain filled the dura completely. Convolutions were seen through the dura. There was no subdural fluid; and on peeling back the dura mater, the pia appeared not to be present, only a tracing of irregularly injected vessels pressed on a smooth brain surface. The pial surface of the brain was dry.



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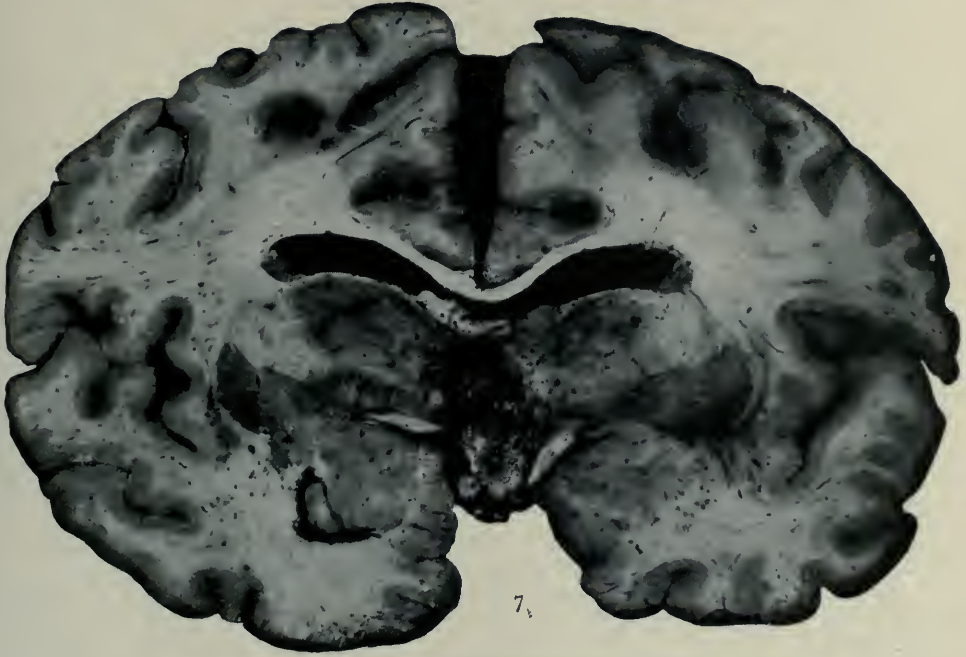

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Plates 6 and 7.—The distention of the third ventricle is more marked; the tumor mass at the base of the cyst now filling the tuber cinereum is larger, and there is a small knob-like extension of the growth on the right. There is marked distortion of the optic tracts. The columnae fornicis lie deeper. The relation to the thalami, and on the right, approximation to the internal capsule which now penetrates farther baseward and is traversed ventrally by the fiber strands from the nucleus lenticularis assembling to form the fasciculus lenticularis (Forel) may be noted. Microscopic preparations were made from the anterior portion of this section.

The superior surface of the brain tended to assume a round form when placed on a flat surface. The hemispheres strained from each other. At the frontal poles, particularly on the left, the convolutions were smooth and pressed out in appearance. The sulci were indicated on the right. The swelling of the brain and the widening and flattening of the gyri were most marked anterior to the posterior lobules. Even the mesial surface showed the flattening of convolutions, especially on the left in the prefrontal region.

At the base of the brain, the vessels were small, the vertebral arteries were unequal, the left being larger than the right. There was some hyalin change in the middle cerebrals, but no sclerosis. The middle cerebral artery on the left appeared very small, and its lumen tiny.

The olfactory bulbs were red and slightly unequal; but the tracts were white. There was a sharp differentiation between the bulbs and the tracts.

The optic chiasm was flattened from pressure beneath, especially on the left side, and the form of the optic nerves was much distorted. The optic chiasm measured a little more than 2 cm. from side to side and spread for a distance of nearly 1 cm. on the left. The tract which leaves the chiasm for the geniculate bodies on the left was broad and thin in appearance from pressure of a swelling from beneath. The distal ends of the optic nerves appeared infringed on and did not assume their round contour, but were irregularly wishbone in shape. The space between the chiasm and the mammillary bodies was translucent gray, it measured 3 by 3 cm., and a puncture of it for securing fluid from the third ventricle showed a thick, purulent, pinkish semifluid substance, a smear from which by Gram's stain, showed many large mononuclear cells with large nuclei; some polymorphonuclears that looked remarkably small in comparison. There were no organisms. When stained by Wright's stain they showed polymorphonuclears (small), red blood cells, and large endothelial and transition lymphocytes. The lobus pyriformis on either side was flattened and thin. The pons measured 4 by 3 cm. and the medulla 2 by 2 cm. The third nerves were involved in a thickening of the pia which forms a part of the floor of the third ventricle.

The optic nerves in their orbital portion showed nothing of note.

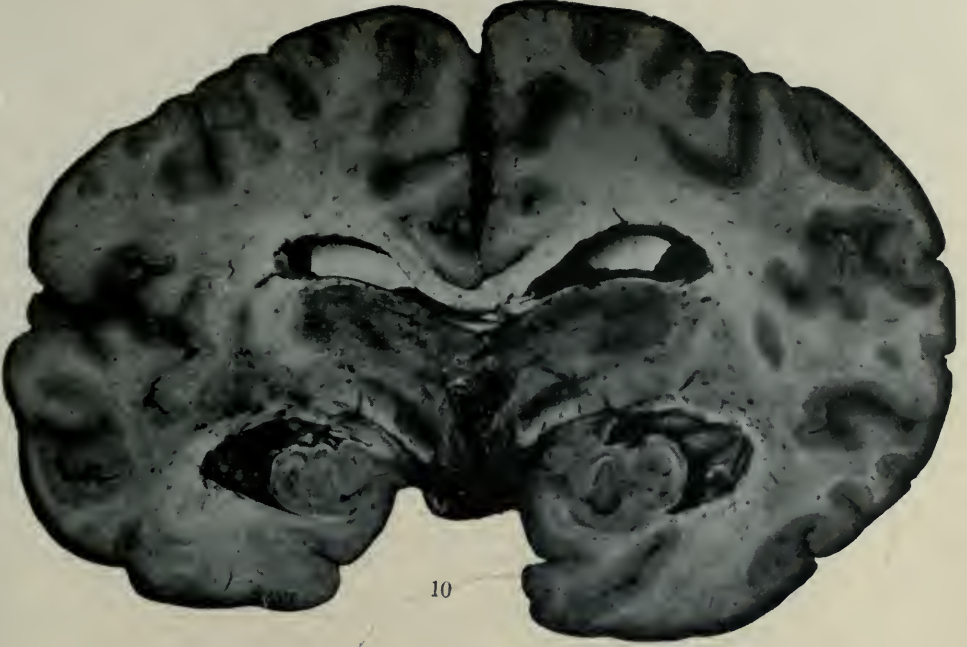
The middle ears, Gasserian ganglions and pituitary gland were negative.

The brain's weight was 1,575 gm. Tigges' formula 8 by 172: 1,376 gm. The gain in brain weight was 199 gm.

The cord was firm and showed nothing of note.

A frontal section revealed a marked difference in size in the anterior section of the lateral ventricles, the right measuring 4 by 3.5 cm. at the wider end, and 1.5 cm. at the inferior portion. The left ventricle cut at the same plane measured 5.3 by 4.2 cm. at the upper, and 1.5 cm. at the inferior. On cross section of the third ventricle, the tissues were found to be soft and edematous, and the capacity of the third ventricle markedly increased.

Anatomic Diagnosis.—The man was well nourished. There were, injection of appendix; sclerosis of the coronaries and aorta; chronic fibrous endocarditis, tricuspid; chronic fibrous endocarditis, mitral; chronic fibrous endocarditis, aortic; fenestration of the aortic valve; apex—scar; pulmonary congestion; chronic interstitial nephritis; chronic perihepatitis; a cyst in the liver. The calvarium was thick; the pia dry; the brain was swollen and smooth. Chronic internal hydrocephalus. Chronic cerebral abscess of the third ventricle and hyaline basal vessels were present. The brain weighed 1,576 gm., the gain in brain weight being 199 gm.



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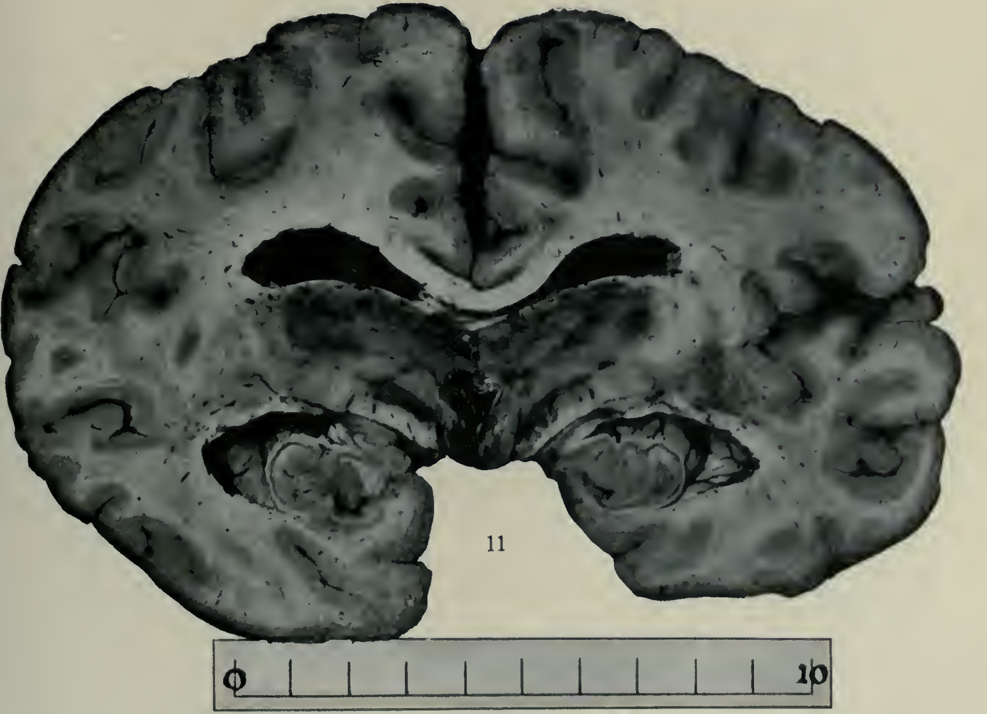

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Plates 10 and 11.—Section passes obliquely through the posterior portion of the massa intermedia and the mammillary bodies. Separating the latter and completely occupying the lower portion of the third ventricle, the cyst is triangular in shape, and there is no growth in the basal portion at this level. The terminal fibers of the fornix are seen entering the lateral nucleus of the mammillary body, and the bundle of Vicq d'Azyr ascending from the mesial nucleus toward the anterior nucleus of the thalamus. Weigert's specimen from the anterior portion of this section shows no loss of myelin in the surrounding structures.

Microscopic Findings.—The visceral organs were fixed in Zenker's solution, embedded in paraffin and stained by eosin and methylene blue and Mallory's connective tissue stain.

Kidneys: There was focal infiltration of the interstitial tissue of the cortex by cells of the lymphocytic series. There was a rare sclerosed glomerulus, compatible with a slight degree of chronic vascular nephritis. They were essentially normal kidneys for a man of 52.

Pituitary: There was normal glandular structure.

Testes: The spermatogenous epithelium showed development of spermatocytes and occasional spermatids, but no mature spermatoza. There was a decrease in the interstitial cells.

Thyroid, suprarenal and pancreas showed no pathologic change.

Spleen: The capsule was thickened. A large amount of intracellular dark brown pigment was present. There was an increase in the connective tissue of the end arteries. Malpighian bodies showed some edema and focal destruction of cells with amyloid deposits.

Liver: Formaldehyd fixation followed by sudan III showed increase of fat in small droplets, chiefly around the portal vessels.

Other organs showed increase in connective tissue around vessel walls. There were a large number of eosinophils in the lymphoid tissue of the large intestine.

The brain and other nervous tissues were fixed in liquor formaldehydi embedded in celloidin, and stained by the methods of Weigert and Marchi, and with cresyl-violet for cell studies.

Longitudinal sections through the heads of the optic nerves showed the disks to be twice the normal thickness, with retention of a definite cupping but deposition of new tissue. There was slight cellular infiltration along the more minute vessels. The retina showed low folds near the disks. There were distention of the vessels within the optic nerves and marked dilatation of the intervaginal spaces with the arachnoid prominent. Cross section of the left nerve showed a small triangular area with loss of myelin.

Peripheral Nerves: They were negative.

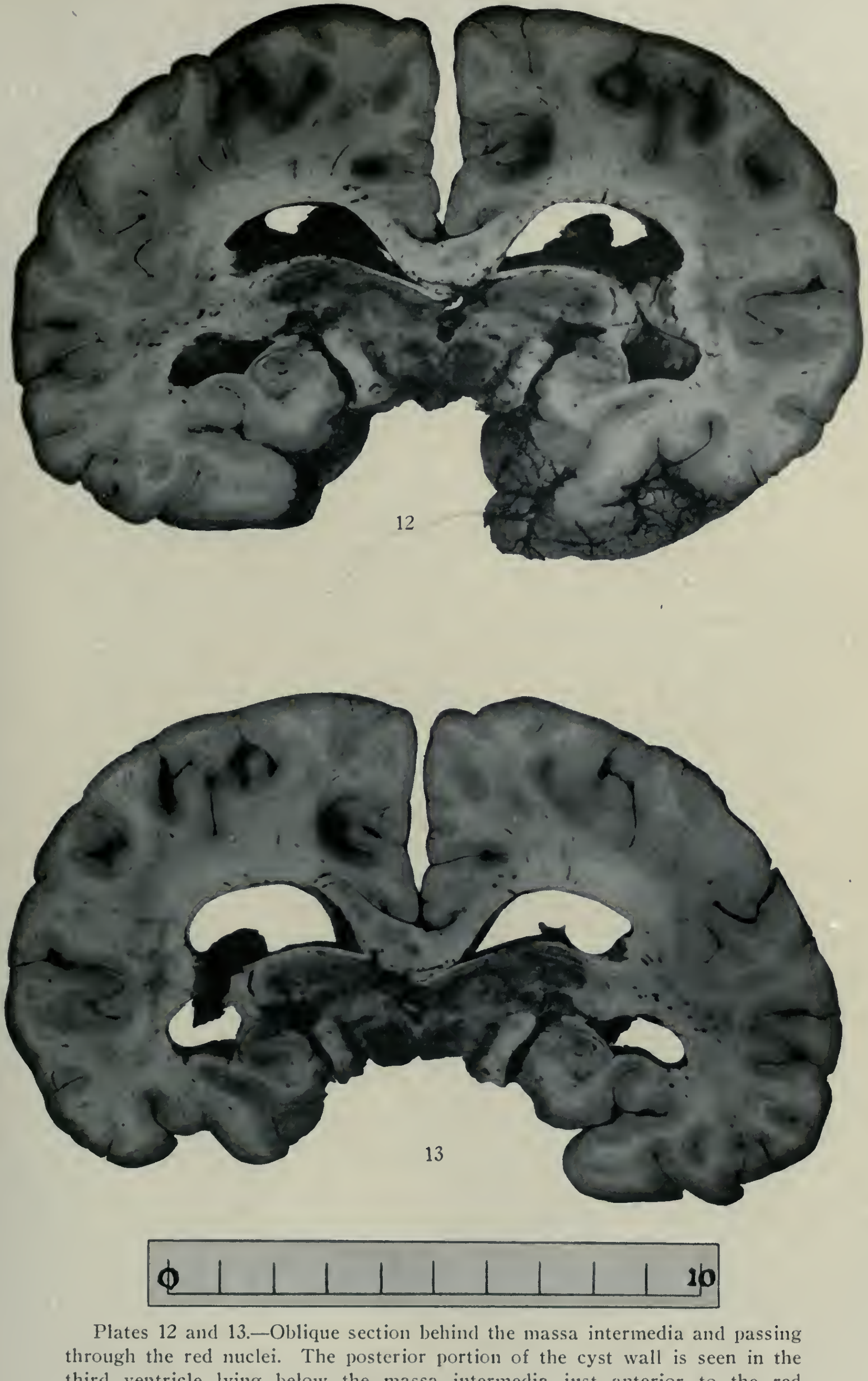
Sympathetic Ganglions: The nuclei were centrally placed. There was peripheral arrangement of the Nissl substance and abundant pigment.

Gasserian Ganglion: The cells were large, and the nuclei were centrally placed. The Nissl substance was finely granular, and evenly distributed through the cells. Few cells showed pigment. The cells were shrunken from the nucleated sheaths.

Cord: Sections from the cervical, thoracic and lumbar regions showed no loss of myelin. A few of the anterior horn cells at each level were filled with fat in very fine droplets.

Sections of the tumor were stained with cresyl-violet, hematoxylin and eosin, Mallory's phosphotungstic acid hematoxylin, Verhoeff's elastica and Van Gieson.

The cyst appears encapsulated by a supporting layer of vascular connective tissue on which is a stratified epithelium, varying in thickness from a few to many cells with extensive desquamation of the superficial layers. There is a tendency to the formation of prickle cells, but no epithelial fibrils or "pearls." Mitotic figures are not numerous. Though the contour of the infundibulum is essentially retained, its tissue is largely distorted by the growth which fills the cavity and which, in this region, appears as a papillary cauliflower-like mass. On the right ventrolateral aspect, there is more abundant vascular connective



Plates 12 and 13.—Oblique section behind the massa intermedia and passing through the red nuclei. The posterior portion of the cyst wall is seen in the third ventricle lying below the massa intermedia just anterior to the red nuclei. There is dilatation and flattening of the third ventricle above the massa intermedia. Sections at this level stained by Weigert and Weigert-Pal show no lesion.

tissue with radiations into the papillae suggesting a tumor stalk. There are several finger-like projections and a small cystic protrusion latterly from this area. In the center of the basal proliferation, there are a few narrow spaces surrounded by a single layer of large deeply staining columnar vacuolated cells resembling goblet cells, and occasionally a few columnar cells with a suggestion of cilia formation. No hair or sebaceous material was found. The stroma is relatively small in amount, contains numerous blood vessels and in some places is infiltrated with polymorphonuclear leukocytes. There are a number of finger-like projections from the tumor but no definite break in the connective tissue capsule was found. The capsule resembles dura in structure and is continuous with the connective tissue about the pituitary gland which was removed intact with the brain. There are a few hyalin globules just below the growth between it and the gland.

There is some gliosis of the surrounding brain tissue, and there is evidence of retrograde metamorphoses. There are numerous collections of polymorphonuclears and endothelial cells containing fat and pigment. Some areas seem to have undergone a hyalin degeneration.

There are none of the large polygonal cells with densely staining protoplasm characteristic of tumors originating from the cells of the pars intermedia. The growth has not the extensive vascularity or angiomatous character frequently seen with plexus tumors.

Sections from the second and third left frontal convolutions were stained with Weigert, Marchi and cresyl-violet. There is a loss of myelin in the fibers of the zonal layer as compared with Campbell's charts. The cortical cells are slightly decreased in number. The remaining cells appear edematous. Frequent cells show eccentric, poorly staining nuclei and diminution in the Nissl substance. This is more marked in the larger cells. Marchi preparations show an increased fat content in a few of the ganglion cells. There is no phagocytosis.

SUMMARY

In this case presenting an epidermoid papillary cystoma involving third ventricle, the tumor probably originated either from a hypophyseal "rest," or as a result of a developmental abnormality of the infundibulum. The clinical signs and symptoms of sixteen months' duration did not lead to a localization before death. Correlation of clinical and pathologic findings has been complicated by the difficulty of separating local from remote and general effects, and the paucity of data as to the normal physiologic function of the structures involved.

It gives me great pleasure to acknowledge my indebtedness to Dr. E. E. Southard for creating the atmosphere that made this study possible and for constant stimulating and helpful suggestions.

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20. In addition to the references already given, the following will be found of interest:

Farnell, F. J.: An Extra Cerebral Tumor in the Region of the Hypophysis, New York M. J. **40**:462, 1911.

Pollock, L. J.: Tumor of the Third Ventricle, J. A. M. A. **64**:1903 (June 5) 1915.

BRAIN TUMORS AS SEEN IN HOSPITALS FOR THE INSANE *

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It is not a rare experience for the pathologist in a hospital for the insane to find at necropsy a brain tumor undiagnosed during life. The objects of this paper are to inquire into the reasons for the lack of diagnosis, and to find out whether the group of brain tumor cases in hospitals for the insane presents any special characteristics as to symptomatology, age or stage of disease on admission, which would distinguish them from cases in general hospitals.

All brain tumor cases coming to necropsy during the past ten years in the Boston, Danvers, Taunton, Westboro and Worcester State Hospitals and the Psychopathic Hospital were studied, as were also single cases at the Medfield, Foxboro, Bridgewater and Northampton State Hospitals. Gummas were excluded. The histories and necropsy protocols of forty-six cases thus collected were studied, and in most instances the brains were examined in frontal sections. In about half of the cases the brains and cords were also studied histologically.

The percentage of brain tumors as compared to the total number of necropsies agrees fairly closely in the different institutions: Boston State Hospital, 1.9; Danvers State Hospital, 1.3, and Westboro State Hospital, 2.6. These percentages are about the same as that found by Cushing[1] in the Johns Hopkins Hospital necropsy records up to January, 109, namely, 1.7 per cent.

The age incidence of patients with brain tumors in the state hospital groups presents an interesting and significant deviation from that of such patients in general. The distribution by decades is: from 11 to 20 years, one; from 21 to 30, three; from 31 to 40, four; from 41 to 50, eighteen; from 51 to 60, thirteen; from 61 to 70, five; from 71 to 80, one; from 81 to 90, one.

The average age is 50 years. Bruns[2] states that brain tumors are most frequent from puberty to 30 years, then from 30 to 40 years; more than half of his cases occurred between the twentieth and fortieth

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* From the Laboratory of the Boston State Hospital. Contribution in a series offered to Prof. E. E. Southard in honor of the decennium of the Bullard Professorship of Neuropathology, Harvard Medical School.

[1] Cushing: In Osler and McCrae: Modern Medicine, Philadelphia, Lea & Febiger **5**:308, 1915.

[2] Bruns: Die Geschwulste des Nerven-Systems, 1907, p. 61.

years. He, however, includes tubercles and gummas, so that the two series are not strictly comparable. In Cushing's series of 130 cases the maximum number occurred between the twentieth and fortieth years. It appears, however, that the average age of patients with brain tumor sent to hospitals for the insane is greater than that of brain tumor patients in general.

The sites of the tumors, in order of frequency were: frontal, fifteen; temporal, six; multiple (metastatic), pituitary and intraventricular, four each; cerebellar or cerebellopontile, five; central fields, parietal and corpus callosum, two each; occipital and peduncular, one each. The large proportion of frontal tumors (33 per cent.) is significant, as in the statistics of Schuster[3] for brain tumors in general, cerebellar growths lead with a percentage of 21.6; followed by multiple, 14.7 per cent.; frontal, 12.1 per cent., and central, 12 per cent.

TABLE 1.—Sources of Group of Patients with Brain Tumor Sent to Hospitals for the Insane

Sent by private physicians (undiagnosed)	31
Sent from general hospitals (undiagnosed)	6
Sent from general and special hospitals (diagnosed)	2
Sent from general hospitals after operation for brain tumor	2
Developed in patients in hospitals for the insane	4
Voluntary admission	1
	46

TABLE 2.—Stage of Disease at Which Patients Were Admitted

Early, that is, soon after the appearance of the symptoms *	4
Advanced, the symptoms dating back from one and a half to three years, and the patients living from one to four years after commitment	8
Greatly advanced, the patients surviving only a few months after admission	19
Terminal	11

* One patient was sent to an institution for epileptics and later to a hospital for the insane.

TABLE 3.—Symptoms Necessitating Commitment

Simple deterioration (neurologic signs also present)	10
Convulsions, with or without mental deterioration	4
Confusion, hallucinations and disturbances of memory, the latter appearing as transitory amnesia, a general reduction of immediate and remote memory, or an almost total loss of immediate retention	9
Lethargy and somnolence	8
symptoms of psychosis (manic-depressive, dementia praecox and senile dementia) antedating symptoms of tumor, but the latter present at time of admission	5
Aphasic and apraxic disturbances	3
Predominantly physical disabilities (blindness and deafness) with irritability and mild paranoid trend	2
Confusion and physical weakness	1

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3. Schuster: Psychische Störungen bei Hirn Tumoren, 1902, p. 298.

The diagnoses made after the patients reached the hospitals for the insane revealed the small percentage of cases in which the correct diagnosis was made and the conditions with which brain tumor was confused. The diagnoses were: brain tumor, fifteen; "organic dementia," eight; cerebral arteriosclerosis, four; epilepsy and paresis, three each; cerebellar disease, two; cerebral hemorrhage, Korsakoff's psychosis, manic depressive and dementia praecox, one each; unclassified, two; senile dementia, two.

MISTAKES IN DIAGNOSIS

The diagnosis of brain tumor was made in only one third of the cases (including four admitted from other hospitals with the correct dagnosis). The cases diagnosed as paresis would probably be correctly diagnosed at the present time with the routine examinations of the spinal fluid now general in hospitals for the insane. Tumors in senile persons are difficult to diagnose. Mistakes in diagnosis tend to fall into three groups: deteriorated cases with neurologic signs are called paresis, Korsakoff's psychosis or put into the catch-all of "organic dementia"; cases presenting focal signs are confused with arteriosclerosis, and those with convulsions are merely called epilepsy.

A surprisingly small proportion of cases of brain tumor are diagnosed correctly. There are several reasons: First, the training and point of view of the physician in a hospital for the insane is psychiatric rather than neurologic, and he is preoccupied with the mental symptoms. In general hospitals, of course, the reverse tendency prevails. Second, the most fundamental reason is that, as a rule, ophthalmoscopic examinations of patients presenting organic signs are not made unless brain tumor is definitely suspected. If such examinations were made, a much larger percentage of tumors would be diagnosed, and until they are made as a routine measure, the percentage of cases of brain tumor diagnosed will not be much raised. It would appear that there is no more reason for neglecting this diagnostic aid in any organic case than for omitting a Wassermann test. Third, the use of the term "organic dementia" as a sufficient designation discourages any refinement of diagnosis. Fourth, the group of tumor cases seen at state hospitals offers peculiar liability to confusion in diagnosis. A large proportion of the cases occur in middle age when deteriorating psychoses, such as paresis, Korsakoff's psychosis and cerebral arteriosclerosis are common, and are naturally first considered. This is particularly true of cerebral arteriosclerosis which frequently coexists with brain tumor; when it occurs, the exclusive diagnosis of arteriosclerosis is apt to be made to cover all symptoms.

A study of the frequency of arteriosclerosis was made in all the available brains of this series. In eighteen of thirty-nine there was notable arteriosclerosis, both of the basal and of the small cortical vessels — all in persons in the fifth decade or beyond. In eight the degree of arteriosclerosis was advanced, and in three small cysts of softening were present.

A microscopic study of the brain was made in nineteen cases. The changes in regions not immediately affected by the tumor were not uniform, nor were they striking, aside from the cases in which arteriosclerosis was present. Lamination was well preserved, but the cells were sometimes askew in arrangement. Chromatolysis was occasionally present, as was an increase of satellites and perivascular pigment. Gliosis, particularly subpial, was frequent, in both the arteriosclerotic and nonarteriosclerotic cases.

Certain tumor groups are of particular psychiatric interest, namely, the frontal, temporal, parietal, ventricular and callosal. These will be considered in detail elsewhere, and only a few points touched on here.

FRONTAL TUMORS

It is difficult to get a clear-cut clinical picture of this group. All of the patients except two (an alcoholic dement and a senile patient) were admitted in the late or terminal stage, and the most prominent feature at the time of entrance was deterioration, usually accompanied by apathy, sometimes however by confusion and excitement, and frequently by somnolence. Deterioration was the most pronounced and constant characteristic. The special importance of frontal tumors for the production of dementia is generally assumed, but has been questioned by von Monakow,[4] Müller[5] and Bruns, among others, their view being that frontal growths usually attain a large size before encroaching on vital centers, and that the psychic changes attributed to the specific involvement of the frontal areas are in reality due to diffuse injury of the cortex, increase in intracranial pressure and interference with circulation. This series is not adapted to throwing light on the question because of the advanced stage at which the patients came under observation, and because of the lack, in most cases, of a full history of the early part of the illness. Nor was it possible always to differentiate between "Benommenheit" and true dementia, although the descriptions in most instances leave no doubt that there was real mental reduction.

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[4] Von Monakow: Lokalisation im Grosshirn, 1904.

[5] Müller, E.: Deutsch. Ztschr. f. Nervenh. **23**:378, 1902-1903; **21**:178, 1901-1902.

Müller,[5] in his analysis of 164 cases of frontal tumor, finds that a larger proportion occur in middle life (from 40 to 60 years) than is the case with tumors of other regions. The maximum of his curve is at from 31 to 40 years, 26 per cent., followed by 20 per cent. at from 41 to 50 years and 16 per cent. at from 51 to 60 years. The distribution of the state hospital series is: from 11 to 20 years, one; from 21 to 30, none; from 31 to 40, two; from 41 to 50, six; from 51 to 60, three; from 61 to 70, two; from 71 to 80, none; from 81 to 90, one. The average age is 50½ years. There are no significant differences between the average ages of patients with frontal tumors and those with tumors of other areas (posterior fossa, 45½ years; all regions except frontal, 48 years). but it is interesting to note that the average age for all classes of tumors is above that given by Müller.

Epileptic convulsions were present over a considerable period in three patients, frontal ataxia in three (causing the diagnosis of cerebellar disease), and in two patients there was early the somnolence supposed to be particularly characteristic of frontal growths.

The large percentage of frontal tumors in the state hospital series may be due, not to any peculiar relation between frontal growths and dementia, but to the fact that disturbances from these tumors are, in many cases, not obvious, unless the patients are under close observation (as they usually are not) until a late stage, when the final deterioration necessitates commitment. Also, in the absence of striking neurologic signs, the psychic symptoms stand in the foreground, and the patient is sent to a hospital for the insane rather than to a general hospital. Another factor, that of an increasing tendency to deterioration with advancing age, will be discussed later.

TUMORS OF TEMPORAL LOBES

Tumors of the temporal lobes are of special interest on account of uncertainty of the diagnostic features of tumors in these regions. Cushing states that, with the exception of the uncinate area, the temporal lobe is a relatively silent region, even on the left. Several authors have tried to differentiate a syndrome common to tumors of both sides. Foster Kennedy[6] emphasizes the epileptic convulsions and their equivalents of dreamy states; crude, subjective sensations of smell and taste, with or without involuntary movements of mastication; and after the attacks, transient weakness of the contralateral lower facial muscles, less often of the arm and leg, and increase of the deep reflexes. The motor symptoms and reflex changes later become persistent.

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[6] Kennedy, F.: The Symptomatology of Temporosphenoidal Tumors, Arch. Int. Med. **8**:317, 1911.

Albert Knapp[7] gives as a syndrome characteristic of both lobes, homolateral ptosis and mydriasis, contralateral hemiplegia and cerebellar ataxia. Bruns and Aswazoturow[8] consider that periods of auditory hallucinosis may be the equivalent of the convulsions. Mingazzini[9] divides the temporal lobe into four zones, the tumors of each having a characteristic symptomatology. Zone 1 consists of the anterior portion of the convex surface, and its syndrome is total hemiparesis, associated with contralateral ptosis and paresis of one or both abducens. Zone 2 comprises the posterior half of the convex surface. In tumors of this region hemiparesis is almost constant, and all branches of the oculomotor may be affected; also, there is a tendency to conjugate deviation of head and eyes and ataxia of the cerebellar type. If the tumor is on the left, sensory aphasia or dysarthria are present with tumors of both zones. In the third zone, the posterior part of the inferior surface, the most frequent signs are unilateral paralysis of the abducens, isolated paralysis of the facial, contralateral ptosis, hemiparesis and hemianesthesia. Aphasic disturbances are frequently absent. The fourth zone includes the anterior part of the inferior-internal surface, and in tumors of this area hallucinations of taste and smell are common.

Arranging the present cases according to Mingazzini's classification, there were two tumors involving zone 1, the first extending beyond the limits of the zone. The case presents so many features of interest both psychiatric and neurologic that it is given in abstract. The patient, a left-handed person, with a left-sided tumor, had also the nearest approach to Witzelsucht of any of the state hospital cases.

Case 1.—*History.*—A woman, aged 35, sent to the Boston State Hospital from a general hospital to which she had been admitted with the diagnosis of hysterical hemichorea, had always been flighty and peculiar. For several years previous to admission she had made no effort to work, but lived on her savings. She complained of numbness in the right hand for two or three years; weakness and tremor of the right arm and leg came on suddenly three months before admission. She had had several slight fainting attacks. On admission she was talkative, hilarious and facetious; her replies were relevant but flippant. She complained of her illness, but was not concerned over it, and showed no insight. She was well oriented for person, place and time; her understanding of surroundings was fair. Remote memory was apparently somewhat impaired; her memory for recent events was good. She would not attempt the educational tests. She had no hallucinations or delusions. She was neat and docile.

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[7] Knapp, Albert: Die Geschwülste des rechten u. linken Schläfe-lappen, 1905.

[8] Aswazoturow: Monatschr. f. Neurol. u. Psychiat. **29**:342, 1911.

[9] Mingazzini: Rev. neurol. **28**: No. 13 (July 15) 1914.

Physical Examination.—The face was expressionless. Movements of eyes and tongue were normal. The right pupil was larger than the left; both reacted well. Vision was undisturbed. There was no choked disk. Hearing was normal, and power of speech was intact. The right arm and leg were spastic and atrophic with marked coarse tremor, increaseed on intention. Knee kicks were increased. Sensation to touch and pain was normal.

Course of Disease.—The patient remained at the hospital for two and a half months. She had alternate periods of drowsiness and exhilaration; during the latter she laughed and talked much of her suffering. She had severe occipital headache. The temperature was continuously subnormal; pulse rate, 80-100; respiration, 20-25. Death occurred suddenly in syncope. No definite diagnosis was made, either at the general hospital or at the hospital for the insane.

Necropsy. — Necropsy revealed a very large cholesteatoma, which had destroyed the anterior half of T1 and T2, the transverse temporals, the lower border of Broca's area, the lower part of the insula, the pyriform lobule, the anterior half of the hippocampus and the subthalamic structures on the left.

CASE 2.—*History.*—The second case was an endothelioma involving the anterior part of the right temporal lobe in a woman aged 57. The illness began two years before death with a change of character, the patient becoming moody, suspicious and irritable and showing defective judgment. Weakness of both legs developed into spastic paralysis; she had general convulsions every three or four weeks. In the hospital she had periods of confusion, alternating with comparative clearness, euphoria and facetiousness; also episodes of auditory hallucinosis, to which she responded energetically. Dysarthria, double ptosis and conjugate deviation of the eyes to the side of the tumor were noted. This case was complicated anatomically with cerebral arteriosclerosis.

The tumor involving zone 2 has been reported by Fuller.[10] The growth was on the left, sensory aphasia was prominent, and auditory hallucinosis was mentioned. The neurologic signs were divergent squint, exophthalmos and bilateral choked disk.

The third zone was involved in two instances. The symptoms in the first patient, a man of 57, were nervousness, irritability, insomnia, possibly auditory hallucinosis, right-sided headache, unsteadiness of gait and tremor. The neurologic symptoms were marked exaggeration of all deep reflexes, general increase in muscular tonus, general tremor, right-sided facial spasm and left Babinski reflex. The tumor involved both the third and fourth zones on the right, extending from the tip to the splenium, destroying also the hippocampus, and extending upward, invading the insula and all structures up to the lenticular nucleus.

The second case is taken up in the discussion of tumor in senile patients.

The fourth zone was the site of a tumor in one instance. The patient was an imbecile of 58, who had deteriorated rapidly in the two years before death. She had olfactory hallucinations, at times calling out continuously, "Oh, that bad smell!" Seven weeks before death she had a convulsion followed by weakness of the right side and involvement of speech. The tumor, a metastasis from a hypernephroma, involved the inferior and mesial surfaces of the left temporal pole.

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[10] Fuller: Westboro State Hospital Papers, Series 1, 1912.

Each of the cases quoted above presents features of the syndromes of Kennedy, Knapp and Mingazzini, but no one case corresponds closely to any of them.

The only instances of pronounced euphoria in the entire state hospital series are the two temporal tumors mentioned above.

TUMORS OF THE CORPUS CALLOSUM

The two patients with tumors of the corpus callosum, involving the entire length of the structure but not limited to it, showed marked mental changes. In the first patient there was increasing slowness of reaction, with somnolence, but his answers were coherent and relevant, and showed comprehension; recent and remote memory were good and school knowledge was retained. Paralysis of the legs occurred late in this disease. The tumor infiltrated laterally into the right frontal lobe. In the second patent the first and the predominating symptom was loss of memory, which progressed until a few months before death there was absolutely no immediate retention. The patient was confused and completely disoriented. Two months before death optic aphasia was noted. The tumor swept out laterally into both parieto-occipital regions. In neither of these cases was there involvement of the cranial nerves.

TUMOR OF THE VENTRICLES

There were four cases in which the tumor (a glioma in each case) was wholly or almost wholly within the lateral or third ventricles. These cases were characterized by profound mental symptoms, in three cases, prominence of the general symptoms of tumor and abundance of neurologic signs. In two cases there was a gradual failure in all mental functions, without active manifestations, and reaching an extreme degree. The third case was complicated by an independent manic-depressive psychosis. The fourth was characterized by increasing lethargy with transient periods of amnesia and confusion, but without true dementia. There was some similarity in the physical signs, the most prominent being unequal pupils, with sluggish reaction to light, in three cases; exaggerated reflexes in three, spasticity in two, tremor in all, and marked ataxia in three cases. Weisenburg,[11] in his article on tumors of the third ventricle, emphasizes the prominence of symptoms of internal hydrocephalus, the marked mental symptoms, the paresis and spasticity, and the ataxia which is present in nearly all cases.

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[11] Weisenburg: Brain **33**:236, 1910.

MENTAL SYMPTOMS

It is fair to assume that because they were sent to hospitals for the insane, the patients studied represent a selected group in which mental symptoms were, to the committing physicians, the most prominent feature of the disease.

The predominance of mental symptoms was due, in a part of the group, to a coincident psychosis of one of the common forms. Thirteen of the patients had been recognized as mentally abnormal—either defective or psychotic—before the development of the tumor symptoms. Three were already in hospitals for the insane, and one was committed after operation for brain tumor. In the other instances the primary psychosis caused the patients to be committed. There were, however, well-developed organic signs in all of the latter cases at the time of entrance. The psychoses represented were: manic depressive, dementia praecox, paranoid condition, senile dementia and alcoholic dementia. In three cases, at least, it seemed to be the development of the tumor that precipitated the active symptoms of the psychosis, and thus necessitated the final commitment.

Excluding the cases in which there was an independent psychosis, with its own characteristic symptoms, and those patients admitted with terminal lethargy and somnolence, in a review of the mental symptoms one is impressed by their undetermined character, the symptoms being most frequently a simple deterioration in all mental functions with apathy, varied occasionally by periods of confusion or slight euphoria. These symptoms occurred with tumors in various situations. Hallucinosis was sometimes mentioned, but was not a prominent feature, except in the temporal lobe tumors. Disturbances of memory were emphasized in the majority of cases — most frequently there was a diminution or even total loss of immediate retention; in other cases, transitory periods of amnesia. The memory disturbances were most extreme in a tumor of the corpus callosum and in one filling the ventricles and associated with an extreme increase of intracranial pressure. In the temporal tumors, as mentioned above, emotional changes were prominent. No pronounced depressions, and no neurasthenic, hysterical or developed paranoid states were found.

It appears that this deteriorating tendency which is so prominent in the state hospital group depends not only on the site of the tumor, but is to some extent characteristic of brain tumors in middle life. Of the twenty cases in this group between the ages of 45 and 60, with no independent defect or psychosis, twelve, or 60 per cent., presented a picture of apathetic deterioration, while of the nine similar cases below 45 years, only three, or 33⅓ per cent., presented a similar picture (two of them frontal tumors). A psychiatric study of a group of young patients with brain tumor in a general hospital would be valuable to contrast with these mature patients. Of the four patients under 30 in the state hospital series, two were imbeciles and the other two were admitted in the terminal stages of the disease. It is precisely these deteriorating, middle-aged patients who would gravitate naturally to hospitals for the insane. This explains why the average age of the state hospital patients is greater than that of brain tumor patients in general.

Schuster goes to considerable lengths in correlating particular mental symptoms with tumors of different regions. We cannot come to any such definite conclusions as he does regarding the association of complex mental reactions with the involvement of certain locations. In fact, the very lack of clear-cut psychiatric pictures is the prominent feature in this series.

BRAIN TUMOR IN THE AGED

There were three tumors in persons 70 years of age or over, the oldest being 86. Only one patient, a man, aged 70, an alcoholic for many years, had symptoms referable to the tumor. On admission he was confused, irritable and suspicious. His answers were incoherent and irrelevant. Recent and remote memory was markedly defective, and school knowledge was entirely lost. There was disorientation for time and place. The physical findings were advanced peripheral arteriosclerosis, emphysema, general hyperesthesia, particularly along nerve trunks, exceedingly active knee jerks and fine tremor of the fingers. The patient had convulsions at intervals of a few weeks during his two years' stay in the hospital. The first of these began in the right arm and leg, but later they became generalized. He complained at first of headache and nausea following the seizures. A year before death aphasia of the motor type appeared with increasing dementia and feebleness. The tumor was a large glioma of the left prefrontal region.

The second case was that of a woman of 72, probably an old dementia praecox, and a hospital inmate for many years. She had had for a long time alternate periods of apathy and extreme excitement, and after one of the latter failed gradually, without special signs or symptoms. The tumor, a glioma, was situated on the under surface of the right temporal lobe, invading also the hippocampus and pyriform lobule. The brain also showed marked arteriosclerosis with small softenings in the thalamus and the lenticular nuclei.

The third case ran its course as an ordinary senile dementia, complicated with chronic nephritis and arteriosclerosis. Mental failure was said to have begun at 75 years. During his nine months' hospital residence, the patient was bedridden, quiet, contented, and completely disoriented for time and place. Recent and remote memory was poor and there was some fabrication. No significant neurologic signs appeared. Failure was gradual. The tumor was a large endothelioma on the orbital surface of the left frontal lobe, protruding into the ventricle. The brain showed the microscopic changes of senile dementia, including plaques.

The reason for the lack of symptoms in some senile brain tumor cases may be that the normal senile brain atrophy lessens the tendency to increased intracranial pressure, and that tumors at this time of life might be expected to grow slowly. In regard to the first point, these senile brains showed flattening of the convolutions only in the vicinity of the tumor and atrophy elsewhere.

It is obvious that brain tumor patients at state hospitals do not in general offer good prospects for operation, on account of the inaccessibility of the majority of the growths, the late stage of the disease, and the frequent complicating factors, especially arteriosclerosis. Nevertheless, decompressive operations would in many cases have added to the patients' comfort. All patients in state hospitals diagnosed as having or suspected to have brain tumor should be examined by a neurologic surgeon, and the possibility of operation considered.

CONCLUSIONS

Brain tumors occur in general hospitals and in hospitals for the insane with about the same frequency.

The group sent to hospitals for the insane is composed partly (30 per cent.) of patients recognized as defective or psychotic before the development of the tumor and partly of those in whom mental symptoms appear with the tumor.

The average age of patients with brain tumor sent to hospitals for the insane—50 years—is greater than the age at which brain tumors usually occur; 68 per cent. of the cases in these hospitals occur in patients between 40 and 60 years.

Frontal tumors predominate, forming 33 per cent. of the cases.

The majority of patients are admitted in the late stages of the disease, and the condition is diagnosed, even tentatively, in only about 25 per cent. of the cases (excluding those admitted to the hospitals with the diagnosis).

The chief reasons for the small proportion of cases diagnosed are that more emphasis is laid on the psychiatric than on the neurologic aspects of the case, that ophthalmoscopic examinations are not made as a routine measure in organic cases, and that, as the majority of the patients are middle-aged or elderly, there are frequently complicating factors, both mental and physical, which would be absent in younger persons.

Cerebral arteriosclerosis is a complication, at least anatomically, in somewhat less than half the cases.

In brain tumor of middle-aged patients committed to hospitals for the insane the usual predominating mental symptoms are simple deterioration and apathy. These were most prominent in the frontal tumors, but were present also in those of other areas, with the exception of the temporal tumors, in which the symptoms were more active and varied. This tendency to deterioration appears to be especially characteristic of brain tumors in middle age.

The development of a brain tumor may be the factor which determines the onset of an independent psychosis in a predisposed person.

In old age, brain tumors may reach a large size without giving characteristic signs or symptoms. The reasons for the atypical course appear to be the senile brain atrophy counterbalancing the tendency to increased intracranial pressure, and the probable slow growtth of the tumor at this age.

It is earnestly urged that the possibility of brain tumor be more frequently considered in insane patients: that ophthalmoscopic examinations be made in all atypical organic cases, and that when brain tumor is diagnosed, the question of at least a decompressive operation be considered.

POSTBELLUM NEUROSES

A CLINICAL REVIEW AND DISCUSSION OF THEIR MECHANISM

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An experience with the American Expeditionary Forces, which comprised a period of intense study of war neuroses at the special hospitals established for their treatment during and after the war, combined with an opportunity to observe the onset of these disorders while serving with a division of troops that had an exceptionally arduous campaign in the Argonne, has increased interest in such cases now appearing at the War Risk Insurance office and at the office of the Federal Board for Vocational Training. It seems worth while to attempt a brief analysis of some of these patients in order to determine what factors are responsible for the continuance of their invalidism at this late date.

THE RESIDUALS OF HYSTERIA

So far the men with the most pronounced symptoms, such as the residuals of a definite hysteria of long duration, have been almost entirely those who served with the British forces and had been in the trenches for long periods before developing grave symptoms. None of these had received prompt treatment in special hospitals near the front, but had been sent back to the home base before active treatment had been instituted, contrary to the method practiced in the American army during the latter months of the war, when special hospitals with each army corps and specialists with each combat division were functionating. In this connection the writer has noted that the majority of the cases from the American army now being seen are in men who developed symptoms during the early weeks of the American campaign before the facilities referred to were available. This tends to show the value of early diagnosis combined with prompt special treatment of any form of functional nervous disease.

The scarcity of postwar neuroses compared with the frequency of neuroses during the war, and the large number returned to duty either at the front or in the intermediate zone while the war was in progress, is an illustration of the value of accurate diagnosis and treatment in the first stages of the disorder. It may also serve as a commentary on our unhappy methods of handling patients with civil traumatic neuroses, so many of whom drift from one doctor to another before the true nature of the trouble is determined, but not before the neurosis has become aggravated and firmly fixed by unfortunate suggestions. To be sure, those who develop traumatic hysteria in civil life have not the same incentive to get well that can be given to the patriotic soldier during a critical period of war when his comrades at the front are stemming the onslaught of the enemy. As our compensation laws are at present framed, those with industrial neuroses have a sordid motive for remaining unfit.

Possibly one might think that any case of "shell shock" remaining uncured a year after the armistice is a case of true concussion of the nervous system, a literal shell shock, with permanent organic change in the brain or spinal cord. This is far from the truth. Such cases are very rare. Many cases attributed to concussion have been seen with permanent mental derangement, but their histories show evidence of mental disorder before entering the service, and their symptoms are those of well known psychoses or of psychopathic states inherent in the individual. Those in whom the symptoms can be called the residuals of organic injury are conspicuous by their rarity. Of a hundred odd cases the writer can recall only two of this nature.

FIVE GROUPS OF POSTBELLUM NEUROSES

The postbellum neuroses now being seen can be classified into five groups: (1) primary neuropathic and psychopathic states, including constitutional neurasthenia, psychasthenia, emotional instability and paranoid states; (2) secondary neurasthenic states, that is, neurasthenic syndromes following influenza, pneumonia or other infections, or following prolonged physical exhaustion and complicated by thyrotoxicosis, myocardial degeneration, tuberculosis or amebic dysentery (but of more importance are the neurasthenic syndromes secondary to a definite mental complex, as will be shown); (3) persons simulating illness or exaggerating trivial complaints and attributing them to military service, usually because they wish to take advantage of the provisions of the federal board for vocational training rather than settle down to the old prosaic job held prior to the war. They feel that the country owes them something because their careers have been interrupted. These might be classified as malingerers or as hystericals, according to the charity of the observer; but, in all fairness, it should be stated that many of these men are entirely sincere in their belief that military service and whatever illness they contracted during service has unfitted them for the work they formerly did, and they are also sincere in their claim that the government should help them to a readjustment. This feeling has been largely fostered by the popular press with its articles on the psychology of the returned soldier; (4) uncured hysterical symptoms, and (5) chronic residuals of true cerebral concussion or other forms of brain injury.

The preceding classification has been made in the order of frequency of type seen in the course of examination of several hundred applicants for either compensation or vocational training. All have had a careful physical examination, as well as a neuropsychiatric examination.

Group 1 represents the sort of man who was overlooked by the draft boards or by the examiners at the training camps and who should never have been accepted in the service. Most of these men have failed to make good even at the training camps or in the intermediate zone overseas, and if they have been at the front they have been quickly sent back with an acute neurosis or psychosis. Now that the war is over, they are of course no better than before; they have much difficulty in adjusting themselves to civil life and are inclined to attribute their difficulties to the effects of some trivial illness or to the strain of military life. A few minutes' conversation with them usually suffices to make clear the fact that the trouble is a constitutional one and not acquired.

Group 2 contains two quite distinct types of acquired psychoneurosis: patients who have definite physical disease as the primary cause and those in whom the cause is a mental complex due to individual circumstances and the patient's reaction to them. Both types are found in men who to all appearances have been normal prior to military service and who have done well in the service up to a certain point. To describe in detail the first type is unnecessary, but attention should be called to the fairly large number of postinfectious states following influenza and pneumonia in which the myocardium is inefficient, and to the cases in which disorder of the thyroid is a prominent feature. As to prolonged physical strain being the only factor responsible for a neurasthenic state lasting for a year or more after the strain is removed, the writer is obliged to be skeptical. Even if no mental cause is found it may nevertheless be the main factor and should be persistently sought. The following case illustrates how a mental complex can be the sole cause of a neurasthenic state:

Case 1.—*History.*—W. C., aged 30, came from intelligent, refined stock and his father had served with credit in the Civil War. The patient had been a successful mining engineer and had never suffered from any nervous symptoms; in fact, he had been particularly apt in mixing with business men and in venturing on new undertakings. He got on well in the training camps and was assigned to a company of engineers sent overseas early in the American campaign. They soon went under fire at Château-Thierry. The patient immediately experienced great fear, and this was intensified when he was thrown to the ground by the bursting of a shell near him. He was not physically injured or rendered unconscious but soon afterward experienced diarrhea and weakness of his legs. He reported to the company surgeon who advised him to go to the hospital. He begged to be allowed to remain in the ranks and as things quieted down he improved. He continued on duty, although not feeling well, and was given light duties. Then his regiment was sent into the Argonne Forest to prepare for the expected advance. The patient carried on with fear in his heart and apprehension as to how he would endure again being under fire, but he would not admit this to any one nor to himself at the time. Again under artillery fire, he suffered extremely from anxiety whenever he heard the sound of shells, and in the intervals he was so weak from emotional strain that he prayed that the next shell might put an end to his misery. Nevertheless, he refused to ask for relief from duty. Fortunately his officers were lenient and sympathetic and spared him all they could by giving him light duty and allowing him to stay in a dugout much of the time. In this fashion he carried on until the armistice, trying hard to repress the knowledge that he was the victim of miserable fear. Shortly after this he was sent to a hospital because of general fatigue, insomnia and mental depression. Subsequently, while in various hospitals he frequently had crying spells and often dreamed of the war, but was loath to talk about it. However, he could not keep from thinking about it and grieved over its horrors and his comrades who had been killed. He was sent to the United States and was under observation for several weeks. His trouble was diagnosed as psychoneurosis and he was finally discharged from the service with this disability on his record. This was a great disappointment to him, because he had planned to reenlist and try to make up for his failure to carry on well at the front by giving more service to his country. After being discharged he tried to resume his old line of work, but when he approached business men he would become tremulous, his voice would fail and confidence in himself would evaporate. So he decided that the war had done something to him which had left him permanently incapacitated for his former occupations and that it would be best for him to prepare himself by special training for work in which he would be on a salary and not obliged to take much responsibility or initiative. He therefore took up the study of geology in order to prepare himself to be an inspector of mines. But he continued to have headaches and insomnia and found it difficult to concentrate and to get through the day without extreme fatigue. Consequently he failed in some of his examinations and felt that his memory was failing. He had planned to join the American Legion but could not bring himself to mix with former service men, although he was particularly interested in returned soldiers who were not well, especially if they had any nervous troubles. He continued to dream often about the war.

Treatment.—When this patient came for examination, apparently he had no idea of the real cause of his difficulties and gave but a meager history. He did not at this time tell about his reaction when under fire. He spoke of his experiences only in a general way, saying that he had been through a great deal and that the war had caused some permanent change in him both physically and mentally. It was only when his war experiences had been gone over in detail step by step, that the above history was obtained and he admitted that he was unable to get rid of a feeling of shame because of his reaction to shell fire, especially since he was the son of a Civil War veteran. He finally was made to realize that this feeling was the cause of his sense of inadequacy, his preoccupation of mind, his dreams and his aversion to discussing the war with other people. After all his symptoms had been linked up with this complex, it was pointed out to him that only by a different and more reconciled and more impersonal attitude toward his reactions at the front could he bring back his old self-confidence and efficiency, and that to attain this mental attitude he must see things in truer perspective; that he had really carried on very well, because in spite of great fear with all the physical and mental suffering that such fear entailed, he had had the moral courage to stay in the game and refuse to be sent back to a hospital until the danger was over. He was told that he ought to feel able to look any man in the face with as much equanimity as any returned hero, and that when he really believed this and felt this way about it, he would begin to get well and would recover entirely. At the end of this talk he showed a marked change in demeanor, his face had brightened, his voice was stronger, his gaze steady and he stated that he agreed absolutely with the examiner and expressed great wonderment that the physician had guessed so well his feelings and mental attitude.

Group 4, presenting uncured or relapsed hysterical symptoms such as paralysis, tremors, amblyopia, nocturnal delirium, etc., contains only five cases, and one of these was complicated by residual symptoms of true cerebral concussion. The other four patients revealed either a conscious or unconscious motive for the continuation of their illness, one of them being undoubtedly quite sincere, and the others showing evidence of conscious simulation.

Case 2.—One of these four patients had been entirely cured of hysteria during the period of hostilities and had been returned to duty and carried on well. After his return to the United States and discharge from the service, having found that his sweetheart had married during his absence, he went on a spree and before he was quite sober reenlisted in the army for three years. A few days later all his hysterical symptoms returned, and he was awaiting discharge from the service on a certificate of disability, when the writer saw him. At this time he had improved very much and presented only an emotional state. He was applying for vocational training.

Case 3.—One of the patients in whom malingering was suspected was a man aged 41, who gave a history of having lost consciousness after a nearby shell explosion, which he claimed had caused a fracture of the skull. This incident had been followed by a long period of apparent amnesia, and he declared that he had been unable to move his left arm and hand since he became fully conscious. He was not treated at any of the special hospitals for neuroses but was sent at once to a base hospital, and from there he was passed along through various hospitals unimproved, being frequently examined and hearing his case frequently discussed. When seen by the writer he was found to be very well informed on the effects of brain injuries. He showed a typical functional palsy of the left upper extremity and claimed to have poor vision, spells of brief unconsciousness and what he called "jacksonian attacks" in his left arm, which he illustrated by a jerking movement of the forearm while being examined. But this was not observed at any other time. When asked to show his tongue it deviated far to the right but at another time to the left. He called attention to a supposed depression in his skull, but examination showed only several small sebaceous tumors in the scalp, and the roentgen-ray examination revealed a perfectly normal skull and brain shadow. This man's attitude was persistently surly and uncooperating. He asserted that he had been told that he would get no better and opposed the examiner's views as to the nature of his trouble and the prognosis with arguments based on what he had observed in patients with head injuries in the hospitals he had been in and on remarks of other physicians. He was opposed to any treatment except an operation on his skull. Finally he was persuaded to enter a hospital and was given daily treatment by physiotherapy and electrotherapy, was handled patiently and tactfully and examined by another neurologist and an ophthalmologist who also assured him of recovery without operation. After a week he had shown no change in his mental attitude, and one day left the hospital without the permission or knowledge of the resident physician, being accompanied by a too sympathetic female relative on whom he relied for financial assistance. In this case we have to consider a true hysteria following a possible slight cerebral concussion, but there are also features in the case that might justify the suspicion of malingering also being present. The idea of being entitled to total and permanent compensation was prominent in this patient's mind, but, on the other hand, he had requested an operation on his skull. Undoubtedly his condition had been aggravated by lack of proper treatment in the early stages of his illness before so many examinations had been made and so many opinions expressed in his hearing.

Group 5.—It is noteworthy that these cases are seldom seen. They present a fairly typical syndrome, consisting of headache, dizziness, especially on change of posture, vasomotor instability and a mental state characterized by complaints of being unable to think quickly or to concentrate, and by excessive and unusual irritability. Furthermore, they give a history of either skull fracture or actual brain lesion from gunshot wounds, followed by immediate unconsciousness, from which they recovered in a few hours, only to relapse again for a brief time. This was followed by a comatose state varied by delirium, which showed a tendency to persist at night for a long time. Many of these patients seemed to recover entirely rather quickly, while others continued to show symptoms even after being returned to the United States. In this connection it should be mentioned that there is such a thing as a "concussion neurosis" superimposed on the syndrome of either a mild or a severe cerebral concussion. Many such cases were seen in France, but it was observed that after a proper explanation of the symptoms to the patient and persistent reassurance as to full recovery, combined with occupational therapy, the disorder cleared up. Regarding the prognosis of more severe concussion cases in which a neurosis is not the main factor, one is obliged to be uncertain until he has followed up the patient for two or more years.

Group 3.—These patients of the third group of the classification require no further comment. Usually they can be persuaded after a careful examination that their claims are unjustified or that their symptoms are not due to military service, and that the best thing for them is to get down to steady work.

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Abstracts from Current Literature

A CONTRIBUTION TO THE PSYCHOPATHOLOGY OF INCENDIARISM. Gustav Bychowski, Schweiz. Arch. Neurol. u. Psychiat. **5**:29, 1919.

The bizarre reactions of mentally diseased persons to incompatible situations are well known. Not infrequently there occurs sudden and persistent writing, tearing of clothes, mimicry, etc. The pathologic aspect doubtless lies in the strikingly inadequate means which are adopted by the individual for the attainment of a more or less conscious purpose. Arson can often be interpreted in that sense. The writer reports the case of a 26 year old laborer of bad heredity who, while in the army, developed an extreme dislike toward one of the officers who had reprimanded him. He immediately generalized his hatred and obtained revenge by stealing a surveying instrument from another officer; at trial he remarked that all officers were of the same stripe. He then began stealing other things—such as railroad time-tables—merely to revenge himself on society in general. Doubtless his antisocial conduct depended in a large measure on a marked enuresis, which made him unwelcome wherever he went. In his downward career, he soon found himself working with a large gang of Italian laborers; here he felt compelled to stay. The situation became unbearable. Without apparent reason he set fire to a pile of telephone poles. Investigation showed that he felt that by so doing he would at once be removed from his present locality. His reactions were typically schizophrenic; the nature of his psychic life, influenced by dissatisfaction, led to a transference of his embitterment, in true infantile manner, to his casual surroundings, which he blamed for all of his misfortune. He lacked the courage to leave his position and search for another; while he realized his own weakness, he blamed it on his surroundings. In burning the pile of telephone poles, he attempted to destroy his entire environment and present reality. It seemed as though the posts for a moment appeared to him to be the symbol of the entire situation, which he could thus destroy most thoroughly by fire.

Another case was that of a housewife, aged 39, who, in the past eight or nine years, developed typical neurasthenic symptoms; treatment in a sanatorium resulted in little benefit. She developed an unfriendly attitude toward other persons. She was dissatisfied with her environment and often threatened her husband should he not allow her to leave the place. A neighbor, who was on the lookout for some fruit thieves, on seeing her pass, remarked in a jocular vein, that she probably was not very dangerous. In response, she stated that she might easily become dangerous enough, and that same evening set fire to his barn. On being admonished by her husband, who told her of the serious consequences this act might entail and that she was in danger of being arrested, she declared that she only wished to get away from her surroundings, and broke down in tears when it was decided, on account of her mental condition, that she was not to be arrested. This wish to get away proved, at closer investigation, to be the index of her entire reaction and the nucleus of the psychic constellation which led to the arson. For three weeks prior to this delict, the patient was daily compelled to see these hated buildings in passing along the street; it was also learned that for eight weeks she felt compelled to carry matches, which she had not been in the habit of doing before and for which she herself could see no immediate use.

The third patient, a man, aged 33, showed definite signs of schizophrenia and was, as well, addicted to alcoholism. One night, thoroughly drunk, he returned home where he was denied admittance. After drinking some more he again returned and was again refused admittance. Having tried several times without success, he looked about for sleeping quarters and saw a shed to which he went. This also was locked and, in a rage, he set fire to it. While attempting to set fire to another shed also, he was apprehended. On examination he was unable to state clearly how it all happened, but remembered that he looked into the flames which appeared so beautiful that he sang and danced as though possessed by the devil; he felt that the fire must put an end to everything and while watching the fire felt greatly relieved of an unbearable oppression. In this case we see primarily the motive of revenge which in a true schizophrenic manner was transferred from the brother-in-law who had refused him admission, to other innocent objects.

The fourth case was that of a 40 year old innkeeper, the victim of psychic epilepsy, who repeatedly came in conflict with authorities for adulterating milk. Although twice punished, he developed a feeling of guilt and when again compelled to submit to an examination for this offense, set fire to his house. The insurance was minimal so that he naturally lost a great deal. He had often felt the compunctions of conscience, which he attributed to persecution by the devil himself, and so was often afraid to go to bed at night. The writer does not feel, on account of the lack of complete amnesia as well as the patient's behavior immediately after the fire, that this deed could be attributed to an epileptic psychic equivalent, but that it more nearly resembled the activity of a psychopath. By suffering the loss of personal property, he could more nearly make adequate atonement for his guilt.

The writer discusses the libido doctrine and does not feel that he can agree with this point of view. The relation between onanism and rubbing sticks to start a fire is not convincing, particularly at the present time when matches are in use, and can hardly be related to coitus symbolism. It is true that fire often appears as an illustration in religious doctrines, for example, that of purgatory, and thus serves as a symbol of cleansing. It is possible that this might apply to the last case discussed. The magic power of fire to free the soul from its captivity in the body and therefore to liberate it for a better existence, developed into the sacred ceremony of the funeral pyre. Doubtless vagabondage, suicide and arson are often dependent on the same underlying misadaptation of the patient to his environment.

The author then illustrates two other cases of reaction types which are somewhat different. The first is that of a highly educated accountant. On account of his exceedingly reticent nature, his free time was spent in taking walks and attending lectures. He was, however, unequal to the practical demands of his work and was reduced in his position. He became greatly dissatisfied and abruptly left his work without a word and without drawing a portion of his salary to which he was entitled. He was quite unsuccesful in every attempt he made to secure another position which he felt society owed a man so well trained as he. One afternoon when his funds were exhausted, he selected some stones and attempted to break a large display window across the street. He did this with great deliberation and it required six stones before the mark was reached. During the course of the trial, he said, that in his opinion society owed him a position; he felt that in prison he could get in contact with the best society, without the expenditure of money that would otherwise be necessary.

The last case was that of a 25 year old man with hypospadia. From early youth he was highly sensitive to his deficiency. The resulting self depreciation became so great that he ultimately attempted to escape from his oppressive surroundings, by developing fugues. In the course of these, he stole money, which was spent in his wanderings.

The author concludes by applying these psychologic methods of individuals, as a means of unloading accumulated affect complexes, to nations. The tremendous movements of nations also offer something which appears irrational, unexpected and therefore secret. Revolutions in particular reveal the double character of necessity and premeditation; while always expected, they are always a great surprise, and exceed not only the expectations of the onlookers, but also those of the instigators. The same is true of war. The onus of an unbearable situation accumulates to the point of discharge; wholly unforeseen consequences and excesses result, despite the best wishes and will of the leaders.

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TRANSPLANTATION OF PERIPHERAL NERVES. G. Carl Huber, an abstract presenting the completion of experimental work on peripheral nerve repair reported in the Archives of Neurology and Psychiatry **2**:466 (Oct.) 1919.

This abstract brings to conclusion the experimental work on peripheral nerve repair reported on in a previous issue. At the time of the former report, in several of the later series, the experiments of long duration had not been brought to conclusion nor had there been opportunity to study microscopically the tissues removed from these and other series. This brief report is thus supplementary to my former report. The series deserving further consideration are especially Series Nos. 11 to 15 of our former report, dealing with stored homo- and hetero-nerve transplants and Series Nos. 16 to 19, dealing with the use of membranous sheaths as wrappings for nerve transplants.

Uniformly favorable results may be reported consequent to use of homo-nerve transplants stored in petrolatum, liquid petrolatum and 50 per cent. alcohol. In each of these series of experiments in animals, kept more than three months after nerve transplantation, functional return was noted in the calf muscles and after four to five months also in the foot interossei muscles. This functional return as evidenced by contraction of the respective muscles on stimulation of the nerve directly, central to the nerve transplant, was checked in sections suitably stained, taken from successive levels of the operated nerves including sections of the regenerated muscle in which new neuraxes within old nerve sheaths were determined. That these new, down growing neuraxes reached the distal nerve segment through the stored homo-nerve transplant was readily ascertained in cross and longitudinal sections of the transplanted nerve segment. The results obtained after the use of stored homo-nerve transplants are quite as uniformly successful as when fresh homo-nerve transplants are used.

The supposition is permissible that in nerves stored in petrolatum and liquid petrolatum at nearly 0 C. temperature, a certain degree of latent viability is retained by the sheath cells and connective tissue cells and the elements of fibrous tissue, though no evidence of the proliferation of the sheath cells could be ascertained, nor of their direct or indirect participation in the down growth of central neuraxes through the transplant. In the case of 50 per cent. alcohol stored nerves, it cannot be supposed that any latent viability is retained by any of the tissue elements of the stored nerve segment. Therefore, there seemed to be justification for the assumption that hetero-nerve transplants stored in liquid petrolatum and 50 per cent. alcohol would serve the purpose of nerve transplants better than would fresh hetero-nerve transplants. Series Nos. 14 and 15 were instituted to test this assumption, and had not been completed at the time of the former report. The results attained may be summarized by stating that in no instance was successful regeneration through a hetero-nerve transplant stored in liquid petrolatum realized. There was noted a marked retardation in the fragmentation of the neuraxes and the myelin in the transplanted nerves, these possibly offering a mechanical block to the down growing neuraxes. In the series of hetero-nerve transplants stored in 50 per cent. alcohol a limited degree of down growth of central neuraxes through the nerve transplant was attained, though by no means as extensive as when a homo-nerve transplant stored in 50 per cent. alcohol was used.

In Series Nos. 16 to 18 of our former report consideration was given to wrapping an auto-nerve transplant and the central and distal suture lines with a membranous sheath, with a view to preventing the encroachment of the surrounding connective tissue and of retaining down-growing neuraxes within a limited field. Cargile membrane in single layers or several layers was used for this purpose. Untreated Cargile membrane was found to be absorbed too early to be of any value. Alcoholized Cargile membrane was found to remain in the tissues unabsorbed for a period of at least five months. In sections through the field of the auto-nerve transplant wrapped in two layers of alcoholized Cargile, and examined approximately five months after the operation, it may be observed that central neuraxes passing distalward in the connective tissue surrounding the nerve transplant are retained within the limits of the Cargile sheath. They are thus guided toward the distal segment of the resected nerve, while the nutrition of the nerve transplant wrapped in the alcoholized Cargile sheath does not appear to be influenced by the presence of the sheath since numerous down-growing, central neuraxes pass through the nerve transplant to reach the distal segment of the resected nerve. With reference to auto-nerve transplants wrapped in autofascial sheaths, it is of interest to note that even in the longer time experiments the autofascial sheath remained clearly defined and without material absorption for a period of nearly a year after the operation. However, their presence in the tissues appear to incite a proliferation of connective tissue within and without the sheath to such extent as to argue against their use in practical surgery. The nutrition of the nerve transplant within the autofascial sheath does not appear to be influenced materially since regeneration through the transplant was attained in all of the experiments observed for a sufficient length of time to allow it. In the series in which a formalized arterial sheath was wrapped about the auto-nerve transplant and the suture lines, six experiments, it is of interest to note that the formalized arterial sheath remained unabsorbed, embedded in the tissues, without inciting undue connective tissue proliferation, for a period of at least six months. The neuraxes that grow distalward are retained within the sheath and thus guided toward the distal segment of the resected nerve.

As concerns the general question of the value of sheaths wrapped about transplant and suture lines, it may be stated that as a general rule these are not to be regarded as necessary, and while perhaps not harmful they serve no specific purpose. Considered in the light of our experimental work, the use of alcoholized Cargile membrane may be justified in certain cases. There is very little connective tissue reaction consequent to their use; they remain in place unabsorbed for at least five months after being placed in the tissues.

G. Carl Huber, Ann Arbor, Mich.

OBSESSIONS. R. G. Gordon, Seale Hayne Neurological Studies **1**:235 (August) 1919.

Gordon takes his text from Bernard Hart's definition of an obsession: "An obsession is an idea, action, or fear, intruding itself into consciousness in a manner which is felt by the patient to be irresistible. It is an incomplete and imperfect thing recognized by the patient as being in itself inadequate and unsubstantial." In the latter half of the definition is embodied the chief point of difference between obsessions and mere memory preservations, dominating ideas either normal or pathologic and impulsive actions. While one may not be in complete accord with the author's explanation of obsessions, yet his classification of these psychasthenic symptoms is interesting enough to bear repetition. In the first group belong obsessive ideas which may be either systematized or diffuse. The former are represented by the so-called pseudo-hallucinations which the patient himself recognizes as unreal and the latter by paralysis of action resultant on inability to "make up the mind." The second group comprises the obsessional acts which again may be systematized or diffuse. The first may come to the surface as "manias" which consist of endless repetition in a vain effort to secure perfection. Whether the particular act is performed or left undone is dependent on the strength of the propelling and resisting forces of the complexes. "Pacts" belong to the same class as "manias" and consist in the performance or avoidance of some action accompanied by a vague feeling of impending ill if the act is not performed or avoided, as the case may be. They are disguised expressions of repressed conflicts. Diffuse obsessional actions are the emotional crises commonly designated "hysterics" and are the motor expressions of incoordinated mental activity.

In the third group are listed the obsessive fears or more familiarly the phobias. Gordon's idea of the mechanism here at work is best elucidated by citing one of his cases. "A patient had a very painful experience in which he was discovered by his fiancée in a compromising situation, although as a matter of fact he was innocent of any deflection from the path of virtue. In spite of his protestations the engagement was broken off, and as a result he no longer cared for his morals and for a time lived a loose life. A year or two later, however, he formed an attachment for another girl, and coincident with the time of his discovery that he wanted to marry her he developed claustrophobia. This fear of closed places was confined to places where he could be observed by others, such as music halls, restaurants and the like. Inquiry elicited the fact, that when he realized that he wished to marry the second girl, the events of the past few years came up before him with painful intensity and he worried greatly as to how they might affect his future prospects. He shirked facing the problem, as he found these thoughts very painful, and tried to forget; in other words, he repressed the conflict unsolved. It is to be noticed that the most painful memory was that of being found in a situation from which he could not escape, for this had wrecked his former engagement, and in order to escape contact with this specific memory he unconsciously detached the dread which belonged to this specific event and reattached it to a more general situation, in which he felt himself observed in a place from which he could not escape. Directly this was made clear to him the phobia disappeared." In other words, according to the author, a phobia results when the emotional halo which surrounds a painful idea is detached and subsequently reattached to an indifferent experience in an attempt to avoid contact with the painful thought.

Whatever may be the faults of Gordon's reasoning concerning obsessions, it is refreshing to note that he is apparently not dominated by the sex keynote of the freudian school.

Strecker, Philadelphia.

THE RÔLE OF THE EMOTIONS IN THE GENESIS OF INSANITIES AND INSANITY FROM THE STANDPOINT OF EVOLUTION. I. S. Wechsler, M.D., J. Abnorm. Psychol. **12**:375, 1920.

The author has performed a distinct service in applying the psychologic principles of William McDougall to the study of the psychoses. He does not reject the abnormal psychology of Freud but properly objects to the attempt made by Freud and his followers to build a system around a powerful instinct, reducing all abnormal mental manifestations to it. The author believes that all the symptoms of "insanity" can be reduced to the simple terms of disturbance in the balance of the primary emotions. He agrees with Morton Prince in that he believes that all sensations and ideas are attached to other ideas and sensations and held in constellations by means of emotional tone which determines the depth and duration of the "clusters." A disturbance of this emotional tone may disrupt the harmony that normally exists between clusters and thus cause conflict which is expressed as psychic disturbance. The author believes that in the normal mind an easy balance is maintained between the various instincts, sentiments and emotions. Every emotion has its opposing emotion, every sentiment its opposing sentiment. If this balance is disturbed the "psychical process" is disturbed. Insanity, therefore, "is a reversal to unbalanced primary instincts and their emotion and sentiments. . . . They seek expression by means of their conative tendency, only there are no opposing emotions to balance them." For instance, "the delusion of persecution may bring out the emotion of fear, without its opposing emotion of anger, and result in cowering, or bring out anger combined with self-assertion, unopposed by fear and subjection and result in positive, active persecutory delusions of the paranoiac." The symptoms of hysteria are produced by an unopposed instinct of imitation, the contrary instinct of self-assertion being absent. These patients have a weak, poorly balanced mechanism for the exercise of their instinctive and emotional reactions allowing some to act unopposed even in response to disproportionately strong stimuli.

The interesting statement is made that the so-called "organic" symptoms of mental disease, such as amnesia, motor or sensory aphasia, are not "insanity" because only symbols are disturbed and not the emotions. No insanity is possible without disturbance of the emotions and the conscious states which accompany them.

The greatest emotional conflict occurs in the evolutionary process that results from the constant striving of the individual against the "species" or "herd." Sympathy is normally a strong innate tendency that ties up the individual to the group. Insane patients have little or no sympathy because only those emotions that strongly satisfy and amplify the ego are found in them. They are concerned only with a struggle against the group and anything opposing this tendency of ego supremacy is destroyed and if the conflict cannot be won, self destruction results as an escape from it. Sex plays a strong part, chiefly in the psychoneuroses, because here occurs the strongest conflict between the individual and the group. The group brooks no trifling with this instinct because it is the means of preservation of the species. As a result the individual loses touch completely with the group to which he belongs and chiefly in dementia praecox "there is a loss of adaptation to reality . . . the emotions remain purely subjective . . . and the result is either disintegration or the reconstruction of the subjective world without reference to objective reality."

Osnato, New York.

MOTOR LOCALIZATION IN THE CAPSULA INTERNA. G. Bergmark. Upsala Läkaref. Förh. **24**:401, 1918.

The classical investigation of Horsley and Beever on localization in the capsula interna of monkeys has also been applied to man and, as we know, their scheme is encountered continually in textbooks and manuals, but no mention is made of the precise information given by these famous investigators as to the different positions of the paths in relation to the knee of the capsule in different horizontal planes. In the case of man there are, as we know, no experimental investigations of this problem. Attempts have therefore been made to prove the applicability to man of the scheme mentioned by means of clinicoanatomic investigations.

Two methods have been used in these attempts. The first consists in the study of degeneration in the capsula interna after limited cortical lesions. In such cases, however, the degeneration in the capsula interna is not confined to certain bundles, but is rather diffuse. This does not militate against the possibility of a motor subdivision in the capsula interna. Mellus found in the monkey, in which we know for certain that there is a motor subdivision of the capsula interna, fairly analogous degenerations in the capsula interna when he had cut out the center either for the thumb or the big toe. He followed the corticofugal paths irrespective of their function. Horsley and Beever, on the other hand, only followed those that could be stimulated electrically. The investigations are thus concerned with two different things. The absence of limited degeneration in the capsula interna in man after small cortical injuries is, therefore, no argument against the possibility of motor subdivision in the capsula interna.

The other method is the study of small lesions in the capsula interna. The occurrence of limited or quite partial monoplegias would be a decided support for a motor subdivision of the capsula interna. The occurrence of capsular monoplegias is, however, a debated question. While, for instance, German clinicians generally recognize this type of paralysis, it is totally denied by Marie and Roussy. Curiously enough, the simple course necessary for the decision of this question, namely, to examine reports of cases of capsular monoplegia, does not seem to have been adopted. The author's examination showed that there was no case of capsular monoplegia to which objections could not be raised. The only case that might possibly be accepted was one given by Dejerine: intense paralysis of the face and, in addition, a slight hemiplegia on account of a small capsular lesion. The practical consequence of this is that brachial or crural monoplegia proves with certainty a supracapsular localization.

This negative result, however, does not definitely disprove a motor subdivision of the capsula interna as one has to distinguish between symptoms of lost function and physiologic localization. We know, for instance, the different cortical centers for the extension and flexion of the fingers, but we never observe cases in which, on account of a cortical injury, the flexion is more injured than the extension. We seem here to have a problem that lies outside the scope of clinico-anatomic investigation.

Author's Abstract.

UN ALTRO CONTRIBUTO ALLA FISIOLOGIA DELL'OCULO MOTORE COMUNE DELL'UOMO. Armando Malagodi, M.D., Riv. di patol. nerv. **24**:99 (June 30) 1919.

The author refers to Luciani's contention and to Bernheimer's experimental work on monkeys as the basis for his belief that the third nerve nucleus has distinct centers for each group of muscles enervated by this nerve. His observation is based entirely on clinical grounds. A third nerve palsy was observed in a soldier who had been injured on the Carso, May 18, 1917, by shrapnel causing a wound 6 cm. long near the lambdoid suture, the bone being exposed. The left cheek and the mouth were lacerated, and there was ptosis with swelling of the lids and some protrusion of the right bulb. There were a divergent squint in the right eye, a left facial paresis and bilateral beginning papilledema. The right pupil was myotic and did not react to light or to painful stimuli; there was no nystagmus. In his discussion, the author makes no anatomic localization of the lesion and does not mention particularly the decreased deep reflexes on the left, nor does he discuss the reason for the left Babinski reflex. There were no sensory disturbances. There was complete paralysis of the right third cranial nerve. This with the left Babinski reflex and the changed left patellar reflex would indicate an anatomic diagnosis placing the lesion at the level of the cerebral peduncles and the exit of the third nerve root fibers, giving a so-called Weber's syndrome. The absence of signs of fillet red nucleus and posterior longitudinal fasciculus injury excludes an injury to the gray matter about the aqueduct or of the central gray matter. The observation, therefore, has only a very doubtful value because the injury was in all probability not to the third nerve nucleus itself. The ptosis had disappeared, August 28, and was the last residual. June 21, the first improvement began in convergence; gradually the pupil began to react to light and accommodation, and finally the ptosis disappeared. From this gradual improvement, the author would draw the following conclusions: First, that there was a hemorrhage in the "central cavity" involving the nucleus of the third nerve; second, that there was no possibility of a root third injury because there was not isolated ptosis; and third, that the lesion must have been a hemorrhage because the gradual absorption explains the regression of symptoms. Why the author leaves out of consideration the undoubted signs of pyramidal tract involvement on the left side and how he explains the exact nature of the lesion or does not explain it rather, is mentioned above. He argues that the respective centers in the third nerve nucleus for each of the eye muscles supplied by it are separated from one another by a considerable distance, the internal recti being closest, the inferior next and the elevator palpebrae last, each of them separate from the other. Despite the fact that the author defends his anatomic findings on purely clinical grounds the futility of such observation must be mentioned.

Osnato, New York.

THE CAUSES, DIAGNOSIS AND TREATMENT OF HYSTERICAL DEAFNESS, WITH NOTES ON THE AUDITORY—MOTOR REFLEX AND THE PSYCHOLOGY OF HEARING. Arthur F. Hurst, Seale Hayne Neurological Studies **1**:279 (August) 1919.

Hurst closely follows Babinski's interpretation of hysteria. Hysterical deafness is, therefore, due to autosuggestion and possibly to heterosuggestion through which agencies the concussion deafness, which often exists at first, especially in soldiers, is perpetuated as a "functional condition." The mechanism is ingeniously but hypothetically explained on the basis of a failure of cortical attention. In other words, the hysterical individual ceases to listen cortically, so he does not hear sounds which reach his auditory apparatus in the usual manner. "In the act of listening the resistance at various synapses in the auditory path becomes diminished by some such process as a throwing out of dendrons, which brings those of contiguous neurons into more intimate connection. In inattention the synapses are unswitched, the resistance being increased by the reaction of the dendrons."

The author comments on the auditory-motor reflex (jumping, blinking, pupillary dilatation following a sudden noise) and believes he has confirmed clinically the experimental observations of Sherrington and Forbes. This reflex is said to be independent of actual hearing. It is a function of the midbrain, the posterior corpus quadrigeminum and the medial geniculate body being closely concerned in its production, and both its afferent and efferent arcs are subcortical. It is abolished in absolute hysterical deafness.

Hurst is rather too sweeping in his disregard of some of the methods which have been customarily used to distinguish between organic and hysterical deafness. History and associated evidence of hysteria are given very little weight because deafness has "been the only hysterical symptom in the majority of cases" and "occurs most frequently in otherwise normal individuals with no symptoms or history of neurosis." These statements are certainly opposed to the usual experience of neurologists. Hurst further insists that hysterical deafness is the one symptom which persists during sleep and in hypnosis. Some significance is attached to the absence of a change in the timbre and intonation of the voice such as is often witnessed in organic deafness. Bone and air conduction give no information beyond either implicating or eliminating the middle ear. Hurst makes his diagnosis rest almost exclusively on vestibular symptoms and reactions as determined by rotation, heat and caloric tests. These are always normal in functional loss of hearing.

The main therapeutic reliances are explanation, with persuasion and reeducation. An extremely interesting case is cited in which deafness of seven months' duration was instantaneously cured by a "psychic" operation. Air and bone conduction and the auditory-motor reflex which had all been absent appeared within twenty-four hours.

Strecker, Philadelphia.

LA CHICHA, BEBIDA DE LOS PRIMITIVOS PERUANOS. Hermilio Valdizan, Rev. de Psiquiatria **1**:62 (October) 1918.

The author says that, according to Bishop Lezanaga, the destruction of the Indian element of the population of Peru is due to the nefarious work of the chicha. The chicha is one of the primitive drinks. It is made chiefly from corn, and is very rich in glucose. There are several different methods of manufacturing this beverage. Originally, it had great religious meaning, and was used at some of the festivals when the people would drink a very great amount of it. Many of the tribes still use the early method of preparation, that is, by the human mastication of the corn, which thoroughly mixes the pulp of the corn with saliva. It is then allowed to stand and ferment. When first made, it has small amounts of alcohol, but as often used, it is buried for a number of years, and with the continuation of the fermentation, it becomes strongly alcoholic.

The author of this article has made a study of the relation of the chicha to the alcoholic psychoses. In a review of the patients in the colony at Magdalena, an institution for the insane, he found that there were hardly fourteen or fifteen patients whose ancestors were users of chicha, and he further states that it is not possible to present a single case of "toxiphrenia" due to the drink of the ancient Peruvians. He does, however, present the summaries of five case histories of patients, who were at the institution at the time of the investigation, in whom chicha could be considered as an etiologic factor, although in none of these cases was he able to show that chicha was the only or the chief cause of the psychosis. In conclusion, he says that the rarity of a "toxiphrenic chichica" in a country which is so essentially a consumer of chicha as Peru, and which has no other asylum than that of the Magdalena, is very suggestive. This allows two explanations: either the chicha is perfectly inoffensive, or the chicha, producing effects on nutrition, acts in a different way from the usual agents of alcoholic toxiphrenia.

A side-light on conditions in Peru is obtained in reading the text, as the author states that use of the straight-jacket is one method of controlling excitements in the asylum.

Solomon, Boston.

ON THE IRRITABILITY OF THE CEREBRUM IN THE EARLIEST YEARS OF CHILDHOOD. G. Bergmark, Upsala Läkaref. Förh. **24**: 419, 1918.

In a child, aged 5 weeks, with hernia of the brain, the writer was able to produce, by means of pressure on different parts of the neck of the hernia, twitchings in different parts of the opposite side of the body. Farthest basally was the point of pressure for the face, above it that for the arm, and highest of all the point for the leg, where twitchings could only be produced however, on one occasion. The twitchings of the opposite side of the body were on the whole very similar, whether one pressed on the right or on the left side of the neck of the hernia. By powerful squeezing of the skin above each point of pressure, it became insensitive to the mechanical stimulation. The case was not investigated anatomically, however, and so it is uncertain whether it was the rind or the projection paths that were stimulated by the local pressure. The writer is, however, of the opinion that the case proves with absolute certainty the irritability of the cerebrum at this time, and bearing in mind our deficient knowledge as to the irritability of the cerebrum in the earliest years of childhood, he thought the case worthy of mention.

Author's Abstract.

MICROSPHYGMIA IN IDIOTS. G. La Roche and G. Richard, Ann. de méd. **6**:75, 1919.

Microsphygmia is a special and generally permanent condition of the pulse independent of cardiac conditions and characterized by difficulty or inability in feeling the arterial pulsations.

This condition was investigated in a series of seventy-two mental deficients of all types, ranging from 4 to 70 years of age.

The condition is most frequent in idiots (twenty out of thirty-nine). In higher mental types, it was found in but one of thirty-three cases (congenital idiocy, twenty out of twenty-two). These findings coincide with those of Richet and Saint-Girons, who regarded the condition as characteristic of idiocy. A frequent concomitant is ichthyosis (30 per cent.). Acrocyanosis was also frequently found.

The pulsation-amplitude is not the same at all places in the arterial supply of the arm. In some instances the radial pulsation being absent the brachial showed nearly normal amplitude. The difference between the radial and carotid or subclavian was more marked. The heart was normal.

The cases were investigated by means of a Pachon oscillometer.

Experiments with amyl nitrite, the oculocardiac reflex, epinephrin and retropituitrin showed that the condition could be altered by vasodilators and simulated by vasoconstrictors. Richet and Saint-Girons had already proved that the condition was not of local anatomic origin (narrowness of the artery) and had concluded that the trouble was of sympathetic origin. The authors confirm this, and add that it is due to a state of hyperadrenalinemia or more generally speaking, a sympatheticotonia.

Kraus, New York.

THE BLOOD PRESSURE IN ANXIETY STATES. G. Euzière and J. Margarot, Progrès méd. **34**:477 (Nov. 29) 1919.

The writers claim that in the so-called anxiety states there is a loss of equilibrium between the vagotonic and sympathicotonic nervous system, with the advantage going to the latter. They have chosen to study especially one feature of the sympathicotonic, namely, hypertension, and they give arterial pressure readings in various groups of cases showing anxiety. Their list is large and comprehensive, but as evidence of possible overelaboration of grouping it should be mentioned that it includes simple melancholias, psychoneuroses, true constitutional anxiety, anxiety psychoses, melancholias with anxiety, anxious hallucinatory psychoses of war, hypochondria and neurasthenia. They consider the elevation of blood pressure frequent in all those conditions; that it is in proportion to the degree of anxiety; that it especially attaches to those showing anguish which is well described as the physical element of anxiety. Briefly, their readings range from maximums of from 120 to 180 to minimums of from 80 to 130, and these figures suggest that all observers might not so readily interpret hypertension from them.

They conclude by saying that their findings have more than theoretical interest and suggest as their application the use of endocrine therapy in all anxiety states. They write: "If the anxiety begins, the anguish is one cause of hypertension; (however) the hypertension can in its turn create the anguish and act as the starting point for the anxiety."

Davis, New York.

COMPLETE SECTION OF THE DORSAL SPINAL CORD DUE TO CONCUSSION OF THE VERTEBRAL COLUMN: CONCUSSION OF THE VERTEBRAL COLUMN (FRACTURE OF THE TRANSVERSE PROCESSES, D 9 AND D 10) WITH CONSERVATION OF THE LEFT KNEE AND ACHILLES JERKS AND THE PRESENCE OF THE REFLEXES OF MEDULLARY AUTOMATISM EIGHTEEN MONTHS AFTER THE INJURY. G. Roussy, M. D'Oelsuitz and L. Cornil, Ann. de méd. **6**:150, 1919.

These authors quote several articles dealing with exceptions to Bastian's law in complete spinal section.

They cite a case of a bullet wound which caused a fracture of the transverse processes of D 9 and 10, without causing any injury of the bodies of the vertebrae or in any way altering the normal configuration of the spinal canal.

This case report is lengthy but extremely interesting. The death of the patient gave the opportunity for a necropsy, and the lesion of the spinal cord is pictured in the article as are sections of the tissues found in place of the spinal cord (about D 10).

The unusual features were the presence of knee and Achilles jerks, on the left. Severe constipation was present. *Complete* absence of the spinal cord for a distance of 1.5 cm. corresponding to D 10 was found. Below the lesion there was degeneration of the pyramidal tract down to L 2. The anterior horn cells were intact in D 12, L 1, 2 and 3.

The case indicates clearly that Bastian's law has very definite exceptions. It is an interesting example of the focal necrosis resulting from concussion without direct injury of the cord.

KRAUS, New York.

CHEMICAL DIFFERENTIATION IN MACROSCOPIC BRAIN SECTIONS. E. LANDAU, Schweiz. Arch. Neurol. u. Psychiat. **5**:68, 1919.

The efforts of the author were directed to the development of a staining method which will give positive pictures rather than the negative pictures usually employed at the present time. The only known stain which will do this is carmin. As is well known, the gray matter appears rose colored, while the white matter remains relatively unstained. Guizzette made use of the chemical reaction, $2\ Fe2\ Cl6 + 3\ K4\ Fe(C\ N)_6 = Fe7\ (C\ N)\ 18 + 12\ KCl$. Berlin blue results. He places a section into a 2 per cent. solution of potassium ferrocyanid for an hour and later transfers this to a 1 per cent. hydrochloric acid solution. It is seen that within a few minutes the globus pallidus is stained from a sky blue to a deep blue color, while the putamen as well as the entire cortex remains unstained. Guizzette concludes from this, that the globus pallidus, the nucleus ruber, and the substantia nigra contain iron salts.

The author conceived the idea of impregnating the cortex and the component parts of the basal ganglia with iron salts, thus staining these structures blue by means of the ferro salt or red by the action of sulphocyanid, according to the formula, $Fe2\ Cl6 + 6KCNS = Fe2\ (CNS)6 + 6KCl$. This is done by placing sections of the brain into a 2 per cent. solution of $Fe2\ Cl6$ for a few minutes to one hour, washing, and then transferring them to a solution of ammonium sulphocyanid or potassium ferrocyanid. Since the red dye of the former reaction is so easily soluble in water, the Berlin blue reaction is to be preferred. The white matter of the cortex remains almost colorless.

The author states that these sections have proved eminently satisfactory for macroscopic demonstrations. He is attempting to apply the same principle to microscopic preparations.

WOLTMAN, Rochester, Minn.

TWO PSYCHOSES CURED BY INFECTIONS. HENRI DAMAYE, Progrès méd. **34**:501 (Dec. 13) 1919.

The author considers that two recent cases in his experience are examples of a theory first expressed by Regis—namely, the supposedly occasional curative effects on a psychosis of a transient infective disease. His first patient, a soldier of 20, after a psychosis of eighteen months' duration recovered normal mental status coincident with recovery from influenza. His second patient made a similar recovery from a psychosis of seven months' duration contemporaneous with recovery from an anginal infection.

The account of each case is brief and one is not told how they were diagnosed, though it is implied that both were considered cases of dementia praecox. The first case in particular, however, seems properly to belong in the manic depressive group. The author does not emphasize the features of diagnosis, but merely states his belief that in each case the concurrent infection was of actual curative value.

Davis, New York.

"EL PSICOANALISIS EN SUS APLICACIONES EXTRAPSIQUIATRICAS." Honorio F. Delgado, Rev. de Psiquiatria **1**:78 (October) 1918.

In this article of thirty-three pages, the author reviews and summarizes the theories of psychanalysis, as expounded by Freud. His description of psychanalysis dates to the time in which everything was related to infantile sexual ideas. He gives a clear, concise and simple account of the freudian doctrine of this period. His review considers a large part of Freud's ideas, including his interpretation of dreams, the psychopathology of every-day life, his ideas of the neuroses and psychoses. Then the author of the article goes on to show how psychanalysis has a value in all fields of human interest and endeavor, especially emphasizing its value in the interpretation of legends and traditions, religious phenomena, language, morals, law, art, philosophy, biology and political economy. All these branches, he says, can be illuminated by the psychanalytic method, and show close relation to the psychosexual life. He concludes by saying: "The things which we have just contemplated bring to the spirit a conviction that we are in the midst of a great revelation. Freud is the explorer of the world of the mysteries of the soul." This article is no new contribution. It is merely the author's presentation of his ideas concerning the freudian psychology. As is quite evident, he is a very enthusiastic follower of Freud.

Solomon, Boston.

LEYENDA DE LA GENESIS DE LOS AMUESHAS (FOLK-LORE INDIANO). Dr. Julio C. Tello, Rev. de Psiquiatria **1**:51 (October) 1918.

"This tradition," according to the contributor, "is of great psychological interest, not only from the general point of view of folk-lore, but from the particular point of view of aboriginal mythology." This myth deals with the development of the worship of the sun and moon among the tribe of Amueshas. It is a very pretty bit of folk-lore, written with a distinctly literary flavor. However, it does not seem to have any especial relation to modern beliefs, nor does the contributor attempt to link it up in any way with psychiatric, neurologic or any broad medical interest.

SIMILARITY IN THE STRUCTURE OF RELATED HEMISPHERES. Zdrawa Jatschewa, Schweiz. Arch. f. Neurol. u. Psychiat. **5**:56, 1919.

The writer attempts to solve the question as to whether the principle of bilateral symmetry of the body can be applied to that of the convolutions of the brain, which, as is well known, show some variations. Occasionally there is almost exact symmetry; in the majority of instances, however, this is not true and it may be hard to discover even a remote similarity in the related hemispheres. On the basis of an investigation of the brains of twenty human beings, eight chimpanzees, two elks, one lion, and four ferrets, he concludes that the principle of bilateral symmetry in the anlage of the convolutions can be demonstrated.

His investigations showed: 1. In some brains the sulci and gyri of both hemispheres are symmetrically developed in one part or another. 2. In that hemisphere where no corresponding superficial gyrus is found, one can demonstrate a more deeply lying gyrus. 3. One may find an excessive growth of a corresponding gyrus. 4. Sometimes opposite convolutions are subject to angulations at a corresponding point; however, this occurs in such a manner that on the one side the direction may be forward, while on the other it is backward.

Woltman, Rochester, Minn.

CONCERNING THE TIME CONCEPT AND A PECULIAR DELUSIONAL CONCEPTION OF TIME IN SEVERE CASES OF TYPHOID. Professor Adolph Strümpell, Neurol. Centralbl. **38**:642 (Oct. 16) 1919.

Our concept of time depends on a fusion of present conceptions with the memories of past events, and it follows that our judgment of time is most accurate when the preceding intervals are short and form an uninterrupted association with the present event. All previous thoughts and experiences have time relations which are dependent on a logical estimate of many differently related circumstances. Of the many factors which may disturb the conception of time, the author cites the influence of the typhoid toxin on four patients, producing a peculiar fixed delusional judgment of time. A girl aged 26 had a high fever and delirium, and after the third week became feverless and rational, other than to insist that she had been in the hospital for seventeen years, that her age was 43, that the last temperature was taken a week before, and other over-estimates of time which persisted for about three weeks, when she suddenly perceived her error.

A woman, aged 40, had a severe case of typhoid and in the ninth week, when otherwise mentally clear, suddenly asserted that she had been sick for two years, considered her age to be 42, was confused about the year, and otherwise magnified the time intervals for eighteen days.

Two other patients showed like disturbances. The author finds it difficult to explain the development and persistence of this delusion, but suggests that the unconscious lapse of time during the delirium, the toxemia, and the protracted stay in bed produced excessive and delusional estimates of time.

Shelden, Rochester, Minn.

ON THE CLASSIFICATION OF NERVOUS AND MENTAL DISEASES. Samuel T. Orton, Am. J. Insan. **76**:131 (Oct.) 1919.

And now comes another classification. Orton appeals to logic, and is not insistent on pigeonholing all cases but on putting a minority in unassailable positions.

Etiology is given first rank as a classifying factor, with groups: (1) infective, (3) toxic, (4) defective and (6) environmental. Group 2, destructive, needs the explanations which are furnished by its subheadings: traumatic, hemorrhagic, anemic, neoplastic. Group 5, metabolic, is complicated and includes errors of food intake and disturbances of endocrine glands not derived from the alimentary tract. The anatomic or physiologic localization is given second place as a classifier, the terms being written against the etiologic subgroups. Clinical entity comes last, instead of first as usual.

So a case is first classifiable as destructive (hemorrhagic), then as encephalorrhagia, and lastly as motor aphasia; or it is first classed as metabolic (dietetic), then as neuritis and finally as beriberi. Manic depressive psychoses and dementia praecox are in the overflow group because the two important classifiers are missing.

This classification, while a valid attack on the standard classification, is not meant by its author to delay the adoption of that scheme by physicians. The very adoption of a first standard makes more easy its replacement by a better second classification, to which Dr. Orton and others are looking ahead.

Bond, Philadelphia.

THE INFECTIVE FACTORS IN SOME TYPES OF NEURASTHENIA.

W. Ford Robertson, J. Ment. Sc. **75**:16 (Jan.) 1919.

Robertson is convinced that chronic bacterial infections are an important element in the etiology of neurasthenia. The nasal passages, the lower respiratory tract, the nasopharynx, the mouth, fauces, the intestinal and genitourinary systems were investigated in sixty-six cases. The *B. influenzae,* the pneumococcus, several strains of streptococci and diphtheria bacilli were isolated and cultured. The logical path of such investigation leads to therapeutic immunization, of which the author writes in glowing terms. For instance, six of the group of seven patients, in whom the *B. influenzae* was discovered, made complete recovery under this form of treatment.

At the present time the question of chronic bacterial infections in the psychoses and neuroses is occupying much attention in this country. There can be no valid objection to what may be termed the bacteriologic method of approach, providing that each study is rigorously controlled and that the conclusions do not exceed the premises. With increasing perfection of technic and conservative, unbiased analyses we may reasonably look for much valuable information.

Strecker, Philadelphia.

DE L'EPILEPSIE LARVEE.

R. Benon, Progrès méd. **34**:435, No. 44 (Nov. 1) 1919.

The writer discusses the question of epileptic equivalents and takes the attitude that, while they occur, they are extremely rare. He believes that the symptom which is fundamental for diagnosis is a lacunar amnesia, which is anterograde, though sometimes slightly retrograde. The writer then emphasizes that such an amnesia is readily simulated, and cites at length a case in which the motive for simulation was to escape punishment for theft.

Davis, New York.

CHEMICAL ANALYSES OF TWO PATHOLOGICAL HUMAN BRAINS.

C. G. MacArthur and E. A. Doisy, Am. J. Insan. **76**:159 (October) 1919.

MacArthur and Doisy report a comparison chemical analysis between two pathologic and three normal human brains. The first specimen was from a paretic; the second from a case of bulbar palsy (?). The cerebellum, the brain stem including medulla, pons, midbrain and thalamus, but more particularly the cerebrum, were studied. The pathologic brains, especially the paretic, one showed a marked loss in lipins (14 per cent.) and extractives. The greatest decrease occurred in the cerebrosids which are closely associated with sheath development. The phosphatids and sulphatids were both lessened. The changes were prominent in the cerebrum, midbrain, medulla and pons, but insignificant in the cerebellum. The clinical diagnosis of "probably bulbar paralysis" in one of the cases must be questioned in view of syphilitic history, a paralytic attack, marked speech defect, assymetric pupils, hallucinations and defective memory. These symptoms seem to be stronger evidence of paresis or at least of cerebral syphilis than of bulbar palsy, particularly when "paralysis of the larynx and laryngeal disturbance" is the only clinical evidence cited in favor of the latter.

STRECKER, Philadelphia.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

J. Hendrie Lloyd, M.D., *President*

Regular Meeting, Dec. 19, 1919

A CASE OF ASTASIA-ABASIA. Presented by Dr. C. W. Burr.

Dr. Burr presented a man, 52 years old, suffering from hysterical astasia-abasia. The patient's early history showed nothing of importance. He had never been a heavy drinker, but had taken some whisky or beer daily for years; he denied venereal infection and his blood Wassermann test was negative. The only nervous shock that he had ever suffered occurred just before his present trouble. He lost $300, all the money he had saved up, by investing it in an unsuccessful junk shop. Immediately after this loss he began to complain of difficulty in walking and in a few days went to bed. On admission to the hospital, Dec. 5, 1919, he was bedridden, not because of a paraplegia but because ataxia prevented standing or walking. On voluntarily lifting his legs from the bed violent ataxic movements started and his malleoli struck together with such force as would cause, in a healthy man, unbearable pain. On the other hand, he had no objective disturbance of sensibility anywhere on the body. On the legs, as everywhere else, he responded normally to touch, pain and temperature stimuli. He localized touch well. There were no motor disturbances in the arms. He had good control of bladder and rectum. The knee jerks were large and quick but not spastic. All other reflexes were normal.

In the left eye there was slight contraction of the field for white and marked contraction for red, blue and green but no reversal. In the right eye there was no contraction for white. For other colors the contraction was the same as in the left eye. The pupillary reflexes and the eye grounds were normal in both eyes. In a few weeks he recovered. The only treatment given was encouragement, with reeducation by making him walk, first with crutches, then with two canes, then one cane and finally without aid.

The case is interesting first, because hysteria rarely appears for the first time so late in life; hysteria is essentially a disease of adolescence and early maturity. Second, when hysteria does begin late in life, ataxia is much more common than hysterical palsies, convulsions and anesthesia.

DISCUSSION

Dr. Francis X. Dercum said that Dr. Burr's patient reminded him of the case of a man whom he had shown at the Neurological Society a good many years ago and in which the etiology was similar. The man was a waiter at a hotel; a fellow waiter robbed him of a considerable sum of money and subsequently he developed astasia-abasia. He was afterward at Blockley where he remained the greater part of a year. Dr. Burr will probably recall a case that Dr. Gilpin showed before the Neurological Society a few months ago; a man who was beyond middle life, developed astasia-abasia after a fall. The symptoms persisted, it would appear, because of the compensation element present.

CASE OF DYSPITUITARISM. Presented by Dr. Frederick H. Leavitt.

A girl, 15 years of age, from the service of Dr. C. W. Burr at the Orthopedic Hospital, at the age of eight months, according to the mother's statement, had had meningo-encephalitis. Since then she had shown evidences of disordered metabolism and physical signs suggestive of a pituitary disorder, as well as palsies and other sequelae of the acute encephalitis. The problem was to ascertain which of her symptoms were due to the endocrine disturbance, and whether the acute infection was responsible for this dysfunction of the pituitary or whether it was a congenital condition.

History.—The history of the case, as well as could be derived from the mother's statement, was: The patient was the youngest of three children. The first was well and married. The second child died at 15 years of age of endocarditis following acute articular rheumatism. The third was born at term by an instrumental and very difficult delivery. She was breast fed and apparently quite well until 8 months old. (At that time the baby was able to stand alone and to take an interest in her surroundings.) She then developed high fever, progressive emaciation and generalized convulsions continuing over a period of three months. When the convulsions ceased it was observed that she was completely paralyzed in both legs and arms. This condition persisted for some months and then power began to return gradually, first in the arms and later in the legs, but she has never been able to walk or to stand since this illness. As the patient recovered the mother noticed that she began to grow fat and that her head seemed to enlarge out of proportion to the rest of her body. At 5 years of age she was very fat and her breasts were very large, almost equaling their present size. The menses started at 12 years of age; they have always been regular, twenty-eight day intervals, of four days' duration and good flow. Pubic and axillary hair started to grow profusely at 14 years of age.

The patient had always been retarded mentally. She had had no teaching either private or public because the mother was told the child never could be taught. She had had no convulsions since her illness. In her diet she preferred red meat, and cared very little for sugar. She always took her tea and coffee without sugar, and she did not care for cake, pie or candy and very little for ice-cream. Pickles and sour things were her preference.

The family history, so far as known, revealed no hereditary taint of mental or nervous disorder. She had had measles at 3 years of age, but no other disease since the convulsive fever when 8 months old.

Examination.—The patient was in bed. She was unable to get out of bed, to walk or to stand, even when her body was supported. Voluntary movements of the hips, knees, ankles and toes could only be weakly made. There was marked flat-foot on both sides. All long bones of both arms and legs were disproportionately short as compared to the trunk. The patient was very fat and of an adiposo-genitalis type. Her head was large and round (circumference 24½ inches), and covered with an abundant growth of well nourished, coarse, dark brown hair. The teeth were very poor in calcium salts. There was considerable fine tremor in both hands, and their coordinated movements were awkwardly performed, probably to some extent the result of the almost complete lack of any training in using her hands. The fingers were short and stubby and there was hyperextension at the interphalangeal joints. At the level of the seventh cervical vertebra there was a false kyphosis. There were no sensory disturbances. The right knee-jerk was smaller than the left but both were much reduced. Stroking the sole of either foot caused at times a fanning of the smaller toes and dorsal extension of the great toe, at other times a plantar flexion of all the toes. Heart action was rapid (112) and regular, the sounds were very much muffled but no murmurs were audible. The blood pressure was: systolic, 98; and diastolic, 60.

Mentally the patient was very deficient. She could neither read nor write, nor even hold a pencil correctly in her hand, and her attempts at copying plain figures were complete failures. She was overly affectionate and amiable and her true mental condition was partly veiled by a silly pertness.

The body measurements were: weight on admission, 138¾ pounds; height, 53 inches; reach of arms, 58 inches. The eye examination was made by Dr. H. M. Langdon, who reported that the visual axes were diverged but could be brought to parallelation on effort. The motions were full and equal, with a tire nystagmus at the limits of lateral excursion. The pupils were 3½ mm. in diameter. There was fair but slow reaction to light, better to accommodation. The media were clear, the disks were oval and quite pale, with distinct loss of capillarity. The margins were well defined. No other fundus changes were found. Diagnosis: suspected primary optic atrophy.

Roentgen-Ray Report by Dr. Ralph Bromer: Examination showed a sella turcica of rather smaller area than normal. Anterior and posterior clinoid processes were somewhat more closely approximated than usual. The tables of the skull were thin. The bones of the hands showed a fusion of the epiphyses with the diaphyses, which usually occurs about the seventh to the eighteenth year. As the patient was 15 years old this was a rather precocious condition. Examination of the teeth showed an unerupted left upper incisor. Deciduous teeth were also present.

Blood Examination: Hemoglobin, 95 per cent.; white blood cells, 8,000; red blood cells, 4,800,000. The blood Wassermann test was negative. The blood sugar test: fasting, level, 0.076 per cent.; after ingestion, level, 0.085 per cent. (sugar tolerance much increased). Carbon dioxid content of the blood: alkalinity was slightly decreased; the alkaline reserve was practically normal—8.4. The coagulation time of the blood was 4 minutes and 58 seconds. Epinephrin test (Goetsch's): Temperature before injection of epinephrin, 98; pulse, 108; respiration, 22; half an hour after injection, temperature, 99; pulse, 116; respiration, 24. The urine after injection showed no glycosuria. Sugar Tolerance: 200 gm. of sugar, ten specimens taken at hourly intervals—a slight trace of sugar was found in the last specimen; 225 gm. of sugar, ten specimens taken at hourly intervals—a slight trace of sugar was found in the last specimen. The urinalysis was negative.

Summary.—From the history, the child seemed normal prior to the onset of the meningo-encephalitis. The weakness of the arms, legs and eye muscles, the tremors and incoordination of the hand movements, the size and globular shape of the head and the retarded mental development were undoubtedly the result of this infection. The symptoms generally recognized as those of a dyspituitarism were the noticeable features of the case; namely, the disposition of fat of an adiposo-genitalis type, the primary sexual precocity and later suppression, her overly affectionate and amiable disposition, the poor calcium metabolism, abundance of well-nourished hair, tachycardia and low blood pressure and the size of the sella turcica, as shown by the roentgen ray. The laboratory findings, especially the increased sugar tolerance, the decreased akalinity of the blood and the slight lymphocytosis, were also corroborative of a pituitary disorder.

Dr. Leavitt said it was impossible to say whether the moderate internal hydrocephalus resulting from the attack of meningitis caused disease of the pituitary, or whether the disorder of that gland was congenital and developmental. The explanation might be that an inflammatory exudate around the pituitary had caused disturbance of its function.

Dr. Leavitt was unwilling in the present fragmentary state of his knowledge to venture an opinion as to whether the anterior or posterior part of the pituitary was most affected, but the clinical picture was that of an hypopituitarism.

A CASE OF CHARCOT-MARIE MUSCULAR ATROPHY. Presented by Dr. George Wilson.

M. R., a woman, aged 56, single, was admitted to the Philadelphia General Hospital in 1914, and is at present on the service of Dr. William G. Spiller. The woman's condition had been present for a great number of years. Her brother said that it began at the age of six and that it had slowly progressed until 1914 when the patient became bedfast and developed incontinence of urine and feces; one month after these symptoms appeared the Philadelphia Hospital was sought as a haven. As far as the brother knew, his sister had had no pain.

The family history was negative; three brothers and one sister and all their progeny were well.

Examination.—The woman was bedfast. Mentally she showed a mild grade dementia. The pupils were contracted, irregular and did not react to light but came down well in accommodation. The optic nerves were normal. The ocular and other cranial nerves were normal with the exception of the auditory nerves, there being a moderate degree of deafness. No speech disturbance was noted.

The hands, forearms, feet and legs were markedly wasted. The hands were in the claw position and the feet were contracted into an equinovarus attitude. The great toes were similar to those seen occasionally in Friedreich's ataxia. The arms, shoulder and pelvic girdles and the thighs were well preserved. The degree of motor power present was in direct proportion to the amount of muscular tissue left. Fibrillary tremors were not observed. The deep reflexes were absent; plantar stimulation produced no response on either side. Sensation was apparently intact in all its forms. There was a considerable kyphotic curve to the spine. The median nerves appeared to be enlarged to palpation. None of the nerves were tender to pressure. The electrical reactions showed loss of reaction to faradism in many of the atrophied muscles.

Dr. Wilson said that in view of the rarity of Charcot-Marie atrophy he thought it well worth while to present the case to the society. Furthermore, the woman had some unusual manifestations. The Argyll Robertson pupil is occasionally seen in this type of disease. Incontinence of urine and feces and dementia are very rare. If the nerves were really enlarged it would make one think of the chronic hypertrophic interstitial neuritis of Dejerine and Sottas; that disease is supposed by some to be a special type of Charcot-Marie atrophy.

DISCUSSION

Dr. John H. W. Rhein said that he was struck with the resemblance that this case showed to the appearance presented by the rheumatoid arthritis. The deformities in the knees, ankylosis of joints, the appearance of the skin, which he had seen very often in rheumatoid arthritis, were very suggestive. There were the curious choreiform tremors of the arm which he had not seen in cases of the Charcot-Marie type of atrophy. It would seem to him that the case was one in which the atrophy was perhaps complicated, it might be with rheumatoid arthritis.

Dr. C. S. Potts said that he had found this woman in the ward several years ago and had examined her carefully. At that time he stated that clinically she appeared to be a case of progressive neuritic atrophy or the Charcot-Marie Tooth type. A history of the time and manner of onset was lacking and not obtainable from the patient, owing to her mental condition. He did not think that Dr. Rhein's idea that the patient had arthritis deformans was tenable. The patient, so far as Dr. Potts knew, had never had pain or tenderness of the joints. There was some ankylosis, but this often occurs when the limbs are maintained in one position for a long time, owing to paralysis or the unopposed action of normal antagonists. It should also be borne in mind that there are changes in the electrical reactions, the atrophied muscles not responding to the faradic current.

A CASE OF JUVENILE TABES WITH AN ARTHROPATHY OF THE RIGHT HIP JOINT. Presented by Dr. Francis X. Dercum.

G. B., a girl, aged 14, a native of Philadelphia, attending school, was admitted to the Jefferson Hospital Nov. 2, 1919, with the statement that she had suffered from an injury to the right hip.

History.—The mother died of tuberculosis; the father was said to be well but an opportunity of examining him was not afforded. The patient did not recall having had any of the diseases of childhood except whooping cough about three years before; in 1916 she had a tonsillectomy. She suffered from influenza in 1918, but apparently had not been very ill. Menstruation occurred February, 1918, but was not established until April, 1919, and had been regular since. She also gave a rather unsatisfactory account of having had "stomach trouble" in May, 1919, for which she was treated at another hospital. Later she said something about having received injections by the veins.

Present Illness: About seventeen days before admission, while taking the class exercise in school and while flexing the trunk to the left, she heard a crack in the hip but felt no pain. She first came under the care of Dr. Rugh in the orthopedic service of the hospital, who made the diagnosis of an arthropathy and transferred her to the ward for nervous diseases. Her condition is at present about the same as on admission. The child limped on the right leg, presented an undoubted ataxia in her gait and also a plus sway with the Romberg test. The knee jerks were absent, the pupils were irregular and fixed to light, the left being much larger than the right.

Examination.—The general visceral examination revealed nothing of significance. A roentgen-ray examination showed a marked distortion of the acetabulum on the right side and irregular deposits of bone salts in the soft parts well below the acetabulum. There was also a fracture through the acetabulum. The head of the femur itself was not involved. The appearance was that of a Charcot joint in which there had been a fracture. The serologic examination of both the blood and spinal fluid yielded a positive result.

Juvenile tabes on the whole is quite rare as are also Charcot joints in patients so young as the one presented. For these reasons Dr. Dercum regarded the case as unusually interesting. The patient is being treated at present by mercurial inunctions and by spinal drainage.

DISCUSSION

Dr. J. Hendrie Lloyd said that he recalled that in the early days of the Neurological Society he brought before it a young girl who was under his care in the Home for Crippled Children. She was afflicted apparently with the form of muscular dystrophy known as the pseudo-hypertrophic type. She had marked atrophic arthropathies of the elbows. They were almost like those seen sometimes in locomotor ataxia. It was before the days of the roentgen ray and laboratory tests. Dr. Lloyd thought it was a novel case because in his observation arthropathies in muscular dystrophies must be rare. The case, as he recalled it, had little about it to suggest tabes, but if such a case were to recur now he would, of course, submit it to the laboratory tests for hereditary syphilis.

A CASE OF HEMIPARESIS WITH CHOREIFORM MOVEMENTS.

Presented by Dr. Andrew H. Woods.

The patient was presented because the involuntary movements of the paralyzed limbs conformed with unusual detail to the type of choreiform movements.

History.—H. B., a man, aged 51, was from Dr. Spiller's wards in the University of Pennsylvania Hospital. Four years before he suffered what was probably a thrombotic affection in some part of the brain, several weeks after which he noticed slight weakness in the right limbs. His eye movements and visual fields were normal. The facial muscles showed no weakness. The tendon reflexes were over-prompt, those of the right limbs being exaggerated. Muscle-tone was increased in the quadriceps extensor, hamstring and calf muscles of the right lower limb. There was a variable patellar clonus on the right; and the right plantar reflex was extensor in type. Power was only slightly reduced in the affected limbs. There was no ataxia. He could thread a needle deftly with his right hand. Light touch and pinprick were perceived promptly in all parts, and there was no spontaneous pain.

There were involuntary movements affecting all parts of the patient's right upper limb, and affecting only the toes and ankle of the lower. These movements ceased when the limbs lay relaxed on the bed; the movements were less marked when his attention was drawn to them, and stopped altogether when he put the parts to voluntary use. After a period of such inhibition, they could be brought out in exaggerated form by causing the patient to fix his attention on the performance of some other act, such as standing on one foot.

Cause of Choreiform Movements.—It would add to the clearness of case reports if the words tremor, choreiform movements, and athetoid movement had standard definitions. The essence of a tremor, as pointed out by Gordon Holmes, is that the affected part oscillated rhythmically, moved by alternate contractions of groups of muscles and their antagonists. Choreiform movements are irregular as to interval, and so have no rhythm; each appears suddenly and passes quickly. Repose of body and mind as a rule quiets them, and they increase with excitement or voluntary effort. But, more rarely, voluntary use of the affected limb will steady it and for a time reduce the muscular contractions. In type they resemble the movements of gesticulation, grimacing and attitudinizing. Athetosis differs from choreic movements in the gradual onset of each individual movement and its slower evolution. A slow tide of tone appears in a group of muscles, rises, then disappears. It may not even produce an overt movement of the part into which the muscles are inserted—the "spasmus mobilis" often described. In type, athetoid movements remind one of the contortions of a person in agony.

The seat of the lesion responsible for the movements in Sydenham's chorea in the large literature on the subject remains a matter of speculation. The optic thalamus is now generally credited with playing an important part on the afferent side in determining the affectual element in sensation; while on the efferent side it possesses centers for the automatic expression of affectual states. The present trend of theory, therefore, prepares us to expect that involuntary movements of the kind now being considered might be due to derangement of this organ. It is unlikely that the causative lesions are irritative, since the resulting movements regularly appear as relatively late symptoms; that is, after the irritative period has passed; and continue indefinitely thereafter. Walshe's contention based on his study of the work of Sherrington and others, seems valid, that the basal ganglions being phylogenetically older than the cortex are probably subject to inhibition from it, but have not control over it. It accordingly seems safer to suppose the lesion is a negative one, that is, a lesion which subtracts the action of some mechanism, and as a result allows some lower mechanism to function without normal control. Holmes' own cases and the review made by him of sixty cases published by others, the experimental work of Sherrington and his collaborators, the recent reports by Wilson and others on lenticular disease, and many cases showing the association of optic thalamus lesions with these involuntary movements, the very apposite study of Rhein of a patient with lenticulo-rubro-cerebello-olivary degeneration, and the findings of many others, all point to the basal ganglions as responsible for athetoid movements, and to lesions in or near these ganglions or their congener, the red nucleus, as responsible for choreiform movements. Tremors have been found associated with lesions in any part between the internal capsule and the medulla oblongata.

As to the exact mechanism, we are left to choose between two general classes of theories: In one class a negative lesion in the tracts connecting the basal ganglions, red nucleus, cerebellum and spinal motor centers disturb a normal balance between the functions of those several parts and that of the corticospinal system. As a result, these complex movements result. In the other class the basal ganglions, particularly the optic thalamus, are regarded as possessing centers whose positive action produced movements fitted to express emotion and which are controlled by the cerebral cortex. In these patients, lesions near the basal centers cut tracts by which the cortical control is normally effected, and thus allow the automatic centers unregulated sway over the muscles involved.

DISCUSSION

Dr. John H. W. Rhein said that he had seen the patient presented by Dr. Woods at the Polyclinic Hospital on a previous occasion. The movements that this patient presented were similar to those in a case reported by Dr. Potts and himself some years ago. Dr. Rhein said that he had not seen the patient but had studied the brain and spinal cord.

This man's symptoms began with weakness and numbness in the legs which never progressed to a great extent. Later there was difficulty in writing, some incoordination of the upper limbs and a tremor, which was to and fro and constant, in the right arm. The knee jerk on the right side was increased. Otherwise, the reflexes were normal, the Babinski phenomenon being absent. The pupils responded sluggishly to light.

At the necropsy, a bilateral lesion of the lenticular nucleus in the putamen was found. In the left putamen, the lesion was in the posterior portion; in the right putamen, it was in the middle portion.

Dr. Rhein stated that he reported a second case of posthemiplegic tremor in a woman of 43 at the Home for Incurables. At the age of 9, following a convulsion, there was a loss of power in the left arm and leg. A clonic contraction of the muscles of the paralyzed members developed. In the hands it resembled pill-rolling movements seen in paralysis agitans. In the legs the movements were to and fro. The reflexes were disturbed very little, the Babinski phenomenon being present.

At necropsy there was found an intact pyramidal tract on each side and a very small lesion in the lenticular nucleus on the left side. Microscopic study showed degeneration of the right ansa lenticularis, the right red nucleus, the left superior cerebellar peduncle, the left dentate nucleus, both inferior olives, the left being totally degenerated and the right to a slight degree, and finally the right restiformis was smaller than the left.

The cases showed what has already been described but not histologically proved, namely, the anatomic connection between the dentate nucleus through the superior cerebellar peduncle with the contralateral red nucleus and the lenticular by way of the ansa lenticularis.

Dr. Rhein believed that this connection, which he termed the lenticulo-rubro-cerebello-olivary tract, constituted the underlying pathologic basis for extrapyramidal motor disturbances. A lesion in any part of this tract, in his opinion, might give rise to a disturbance of motion of the extrapyramidal type. The case was somewhat confirmatory of the position taken by Dr. C. K. Mills, who explains motor disturbances through the intermediation of the cerebello-rubro-thalamo-cortical apparatus and the cerebro-rubro-spinal apparatus, asynergic phenomena resulting if the cerebellar influence is withdrawn, hypertonicity and involuntary movements if the tonectic excitation from the striatum is withheld.

Most hemiplegic movements such as tremors, choreiform movements and the like have been described in cases in which lesions have been found in the thalamus with or without involvement of the caudate nucleus.

In some cases, the lesion is in the crus-cerebri; in others, in the cerebellum and in still others in the superior cerebellar peduncle, red nucleus and tegmentum; in others the cortex has been implicated, and finally in some cases the cord is the seat of the lesion.

Various theories have been advanced to explain the origin of the movements. Some advance the theory that the optic thalamus is the coordination center. Another theory is that the corpus striatum inhibits movements and that the thalamus organizes movements, and that a lesion of the corpus striatum produces lowered inhibition. The theory that Charcot advanced that there was a chorea bundle has long since been abandoned. The theory that it is due to an irritation of the pyramidal tract is unsatisfactory. It seemed to Dr. Rhein that the explanation of these movements was the one that he had given above; namely, that the automatic movements were due to the implication of the tract that leads from the lenticular nucleus to the dentate nucleus, and then from this to the inferior olives.

Dr. Charles M. Byrnes said the case was especially interesting in regard to the paresis of the facial nerve and he hoped that Dr. Woods would have more to say about the degree to which the facial nerve was involved. Was the whole nerve at any time completely affected? A recent paper has appeared in the *Revue Neurologique* which explains the escape of the upper part of the face in upper segment lesions of the facial nerve on the belief that the fibers to this portion of the seventh-nerve reach the nucleus by way of the ansa lenticularis. If this be correct, and if the lesion in Dr. Wood's case is located in the basal ganglions, it would be interesting to know whether the upper facial branch had been involved. Huntingdon's chorea has also been attributed to a lesion in the lenticular nucleus, but Dr. Byrnes did not recall an instance in which this disease has been associated with involvement of the facial nerves. It is, of course, probable that a lenticular lesion might be present in Dr. Woods' case without involvement of this supposed path of the corticonuclear facial fibers.

Dr. William G. Spiller said that the movements in this case were distinctly choreiform. In most of the cases of so-called posthemiplegic chorea the movements are those of athetosis.

In referring to the subject that Dr. Byrnes had brought up, namely, the escape of the upper branch of the facial nerve in central facial palsy, Dr. Spiller said he was observing at that time a case of lethargic encephalitis, which is a form of acute bulbar palsy, and the man had complete loss of power in the lower facial distribution of each side but could close the eyelids of each side. It is well known that in progressive bulbar palsy the upper part of the face is less involved, although the lower part of the face may be completely paralyzed. It is improbable that the nucleus of the upper division of the facial nerve is remote in the brain stem from that of the lower division. Dr. Spiller would attribute this greater escape of the upper branch to the fact that the nucleus of this branch receives much stimulation from both sides of the brain, so that a lesion that might paralyze the nucleus of the lower branch might have less effect on the nucleus of the upper branch, which probably has a greater vitality and is more resistant on account of its double innervation from the cortex. This is not quite the same idea as that employed in the explanation of the escape of the upper branch in supranuclear lesion, when one corticofugal path to the nucleus is affected while the other escapes.

If the nucleus of the upper branch is a part of the general facial nucleus in the pons it must be a distinct group of cells, much as the nucleus of the fibers to the anterior tibial muscle is distinct from that of the fibers to the other muscles in the external popliteal nerve supply. In acute poliomyelitis the anterior tibial muscle may be the only one in the external popliteal supply to escape, or it may be the only one in this supply affected, showing that the nucleus of its nerve supply must be somewhat apart from that of the nucleus of the remaining supply.

Dr. Charles K. Mills said that the case was interesting in connection with the consideration of the cerebral tonectic apparatus and mechanism, a subject to which he had given much attention. In the patient under discussion he thought it probable that the lesion was one of the lenticula, as somewhat similar cases had been reported by von Monakow and others. Years ago, while investigating the pathology of chorea, Flechsig observed small globular bodies on the vessels in the middle and inner zone of the lenticular nucleus. These bodies were supposed by Flechsig and others probably to be the result of calcareous degeneration of some new growth whose nature had not been determined. Dr. Mills believed that both the premotor cortex and the striate body were portions of a cerebral apparatus concerned with maintenance and distribution of tone. One of the special functions of the lenticula seemed to be the rhythmitization of synergic movements differentiated in the cortex and controlled in part by the cerebellum. Such rhythmitization is interfered with in chorea, or at least in certain types of choreic cases.

Dr. Charles S. Potts said that Dr. Rhein had mentioned a case that he had studied pathologically with Dr. Potts. It was so long ago that Dr. Potts did not recall the entire history of the man, but he remembered his movements were very similar to those of the man presented by Dr. Woods.

Dr. A. H. Woods, replying to Dr. Byrnes' question, said that the patient showed no facial weakness, and apparently the facial nerve was not involved at any time. From the paucity of symptoms, one would judge, the lesion was small, so that a tract in or near the claustrum might easily have been spared.

A SPECIMEN FROM A CASE OF CEREBELLAR TUMOR. Presented by Dr. P. G. Hamlin.

History.—A negro, a man, aged 40, was admitted on Oct. 11, 1919, to the Philadelphia Hospital, service of Dr. C. W. Burr. The patient complained of pain in the head, pains throughout the body, soreness and stiffness of joints and dizziness; the onset of his illness was four months previous to his admission to the hospital. He had had a chancre fourteen years ago and had been a daily drinker of whisky. He had had no difficulty in walking until just before coming to the hospital.

Examination.—Station was poor, worse with the eyes shut. The gait was slow and careful, as if in fear. When he was in bed, no ataxia on voluntary movement of the legs was present. There were no cranial nerve palsies. The hearing was poor but had been so for years. The pupils were equal, the right pupil reacted fairly well to light, the left sluggishly; both reacted to accommodation. No nystagmus, no hemianopsia nor disturbance of speech, either paralytic or aphasic were present. In the hands there was no adiadokocinesis. The biceps jerks were normal on both sides, the patellar jerks were exaggerated on both sides. The Achilles jerk was exaggerated on the right, normal on the left. There was no ankle-clonus. Bladder and rectal control were normal. No muscular wasting in arms or legs was present and motor power in both arms was good. Far advanced pulmonary phthisis was found. The mental state was good.

Nov. 7, 1919: The man had lost all motor power in his legs, he could move the arms in all directions, but weakly. A reflex was not brought out on stroking the outer side of the sole but on stroking the inner side there was a slight withdrawl of the foot. Marked intention tremor of both hands with slight tremor of hands at rest was noted. The cremasteric, abdominal and biceps jerks were normal on both sides. There was marked general emaciation of the whole body, but no local muscular atrophy. Rigidity of the neck had developed. Both pupils were dilated, the left was larger; reaction to accommodation was better than to light. Incontinence of the bladder and rectum was complicating the scene. At the beginning of an examination he answered well, but in a few minutes he became confused.

On admission, the temperature was normal. Two weeks later it rose to 100 F. From then until death it ran from 101 to 102 and immediately before death it rose to 106.2 F.

During his stay in the hospital he complained constantly of headache, especially over the right eye. He had not vomited and complained little of vertigo.

Blood Wassermann: Cholesterin, + + + +; acetone, + +; syphilitic liver, + +. The spinal fluid Wassermann test was negative. Cytologic examination of the spinal fluid showed 250 cells per c.c. The percentage of large lymphocytes and polynuclears was equal. Endothelial cells were present; no bacteria.

Necropsy.—On the under surface of the right lobe of the cerebellum a tumor mass about 4 cm. in diameter was observed. Over the mass the dura was densely adherent. The tumor was flattened out from above downward, well outlined, and dense. Much of the cerebellar substance was encroached on where the tumor mass fitted in. The cerebellopontile angle was not invaded by the growth. Subsequent histologic study of the tumor and staining for spirochetes by the method of Levaditi led to the diagnosis of tuberculoma. The slides were studied by Dr. Baxter L. Crawford and the diagnosis was concurred in by Drs. Coplin and Rosenberger.

Complete Pathologic Diagnoses: Tuberculoma of the right lobe of the cerebellum; extensive bilateral tuberculosis with multiple cavity formation of the left lung; tuberculous lymphadenitis, mesenteric; emphysema, right lung. The heart showed myocardial degeneration and the kidneys moderate acute diffuse nephritis. Chronic adhesive bilateral pleuritis was present.

One interesting feature in this case is that the tuberculous condition so masked the cerebellar manifestations that the presence of the cerebellar new growth was not recognized during life; in fact, tumor was not thought of. Exact diagnosis would in any event have been impossible. The mental state was probably due to the general infection. It, of course, was not caused by the tumor. Another interesting feature is the concurrence of syphilis and tuberculosis. Circumstances prevented a fundus examination, and the condition of the optic nerve head is therefore unknown.

LANTERN DEMONSTRATION OF INTRACRANIAL FOREIGN BODIES. Presented by Dr. Samuel D. Ingham.

Dr. Ingham gave a lantern demonstration illustrating the radiographs of a series of cases of intracerebral foreign bodies occurring among the soldiers with head wounds who were under observation at U. S. Army General Hospital No. 11, Cape May, N. J., from October, 1918, to July, 1919. Radiographs were made as a routine measure in all cases of gunshot wounds of the head, from exposure both in the anterior, posterior and the lateral aspects of the cranium. This means practically insured detection of foreign bodies, and determined with a fair degree of accuracy their location as well as the point at which each penetrated the skull. These cases were of neurologic interest on account of the focal cerebral symptoms manifested, and also as illustrations of the great tolerance of the brain to the presence of foreign bodies within it. The salient clinical aspects of the cases illustrated were briefly outlined, and the point of entrance, course and position of the foreign body in each case demonstrated. It was considered, however, that cases of this type were not particularly favorable for the study of cerebral localization because of the wide extent of the lesions usually produced by the deep penetration of the foreign bodies.

Among 200 cases of head wounds the presence of intracerebral metallic foreign bodies was determined in twenty-three, and the cases illustrated were representative of the group.

It was noted with interest that the presence of foreign bodies within the brain did not appear to cause any symptoms, with the one exception in which jacksonian attacks were evidently related to the presence of a small piece of shell just beneath the motor cortex. There was no evidence in any case that a foreign body within the brain changed its position through the influence of gravity, and no late abscesses of the brain developed as might have been expected as the result of infection being carried deep into the substance of the brain. This type of case was, however, treated with much respect by the surgeons, and it was the rule not to operate for the repair of cranial defects on patients with intracranial defects.

DISCUSSION

Dr. William G. Spiller said that two of the cases reported by Dr. Ingham were particularly interesting, those in which the wound of entrance was in the frontal bone. In one of these cases the man was unable to find his way about. Dr. Ingham did not speak of the loss of orientation in the other case. Dr. Spiller said he was interested in this subject, because Marie and Béhague recently had reported the loss of orientation in space but not in time resulting from a lesion of the frontal lobe. Dr. Spiller had not been able to establish the correctness of the interpretation. He had had a patient with left frontal lobe cyst resulting from a fracture of the skull and that man traveled fifteen miles alone to Camden after he had been injured. This was a clear indication that he had not lost the orientation in space.

Dr. Ingham said that of the entire series of head wounds observed at Cape May there had been a number that showed mental deterioration, and those in which the dementia was most marked had lesions involving both cervical hemispheres. The case about which Dr. Spiller inquired was the only one in which there was a definite disorientation for place, but other symptoms were also prominent; for instance, loss of memory was such that the patient did not remember having been in France. The lesion in this case involved the right frontal lobe and extended diagonally backward and across to the posterior part of the left parietal lobe. In three other cases with dementia the lesions involved both frontal lobes; in two of these the mental symptoms closely simulated paresis. In contrast to these patients having bilateral cerebral lesions were many other cases of unilateral wounds with extensive loss of brain tissue from all the different parts of the cerebrum, including the frontal areas, but which presented no definite evidences of mental deterioration. In Dr. Ingham's experience he had not been able to formulate any definite idea as to the cerebral localization of psychologic symptoms. Bilateral cerebral lesions, however, and especially those involving the frontal lobes, appeared to cause much greater mental disturbance than wounds of similar size affecting only one hemisphere.

Book Review

KOMPENDIUM DER TOPISCHEN GEHIRN UND RUECKENMARKSDIAGNOSTIK. KURZGEFASTE ANLEITUNG ZUR KLINISCHEN LOKALISATION DER ERKRANKUNGEN UND VERLETZUNGEN DER NERVENZENTREN. Robert Bing, a. o. Professor an der Universität Basel. Vierte, neu durchgesehene Auflage. Mit 97 zum Teil mehrfarbigen Abbildungen.

In his preface to the first edition (1909) the author states that his aim is to present a terse, practical vade mecum for the general practitioner and surgeon who may turn to it when confronted with the task of localizing some pathologic process of the central nervous system. He also undertakes to simplify what is generally considered a complicated domain.

That one untrained in neurology could make any desired localization with the assistance of this book, may well be questioned; but the author has done his work well. Except for considerable diffuseness of style, a sort of conversational excess apparently carried over from the class room, it is hard to see how he could have got more into the 222 pages of text. The subject is treated methodically, and the sequence is quite as logical as necessary conditions permit.

First, there is a sketch of the structural anatomy of the cord, followed by an outline of the essentials of cord physiology. Then are considered seriatim the symptoms indicating involvement of the lateral columns (pyramidal tracts), the anterior horns, posterior roots, posterior horns, etc. Next are taken up the symptoms of a transverse lesion at different levels and of hemisection. This is followed by a detailed presentation of motor, sensory and reflex innervation from different cord segments, which is especially comprehensive as regards the muscles.

Chapter 3 takes up the symptom-groups of high and of low cord lesions and should enable even the neophyte to distinguish one from the other. Eighty-seven pages, about two fifths of the text, are devoted to the spinal cord, the remainder to the brain. Peripheral nerves are not considered.

Consideration of the topographic diagnosis of brain lesions begins with sixty-eight pages on the brain stem (medulla, pons, crura). It includes structure and physiology, and symptoms of lesions of this complicated region, including the cranial nerves. Nothing of importance is omitted; the conclusions are in general sound and the matter is presented about as clearly as could be done within the limits set by the author.

Lesions of the cerebellum include seventeen pages. On this somewhat uncertain ground the author is perhaps a bit too sure; a few statements are to be taken with a grain of salt. But the essentials of cerebellar diagnosis are well outlined and the remarks on vertigo and nystagmus will be helpful to those not neurologically trained.

The final fifty-three pages contain an outline of such cerebral anatomy and physiology as are directly applicable in clinical diagnosis: the general meaning of the principal sensory and motor signs; the localizing significance of the various central disturbances of vision, speech, hearing, smell, intelligence and character.

Obviously, this book is the result of a great deal of honest, earnest work. It contains an enormous amount of information, and if the author had the gift of clear, succinct statement, it would be almost without fault.

The illustrations are good because they are clearly indicative and therefore helpful. They might be increased with benefit to the beginner, especially in the section on brain stem. Especially do we commend the diagrams of transverse sections of the extremities. The index is not of the best, but it is good.

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AN ACCOUNT OF THE WITCH CRAZE IN SALEM, WITH REFERENCE TO SOME MODERN WITCH CRAZES *

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PHILADELPHIA

Belief in witchcraft and the prosecution and execution of those accused of it have existed in all countries from the earliest times. Even now the belief is prevalent in certain parts of this state; and it is especially prevalent among the negroes of all countries. In the early days of this country such a belief seemed to be especially prevalent in the New England colonies, the first case of witchcraft occurring at Springfield, Mass., in 1645. From that date sporadic cases appeared from time to time until 1692, when the epidemic of accusation in Salem, Mass., and its vicinity occurred. During a period of four months, 250 persons were accused and put in jail. Of these, nineteen were hanged and their property confiscated. Two died in prison from exposure and bad treatment, and one was pressed to death, a pleasant mode of torture, which will be described later. It must be remembered that this represented a rather large percentage of the population at that time.

For much that is to follow I am indebted to an interesting book, entitled "Witchcraft in Salem Village," by W. S. Nevins.

ORIGIN OF WITCH CRAZE IN SALEM

The craze had its beginning in the home of the Rev. Samuel Parris, the pastor of the church in Salem. His family consisted of a daughter aged 9, a niece, Abigail Williams, aged 11, and a servant, Tituba, who was half Indian and half negro. She and her husband had come from the West Indies where belief in witchcraft was very strong. Associated with these were four other girls—Ann Putnam, 12 years old, the daughter of Thomas Putnam; Mercy Lewis, 17 years of age and a servant in his family; Elizabeth Hubbard, 17 years old and a niece of the village physician, and Sarah Churchill, aged 20, a servant in the family of George Jacobs, Sr., who, with his wife, was afterward

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* Presidential address delivered before the Philadelphia Psychiatric Society, Jan. 8, 1920.

accused. Mercy Lewis at one time had lived in the family of the Rev. George Burroughs, who was also accused. Several of these persons belonged to prominent families. It will be noted that some or all of them figured in all the trials, although as time went on others also became associated.

It is said that for purposes of amusement the West Indian servant used to practice tricks and incantations common among the natives of her home. During these performances they did strange and unusual things, such as getting into holes, creeping under chairs, assuming odd postures, performing various antics, making odd gestures and uttering foolish, ridiculous speeches. While it is natural for children to do such things more or less, they were not countenanced by their Puritan relatives and masters. Great, therefore, was the consternation when they learned of it. Dr. Griggs, whose niece was one of the participants, was called, and not being able to make a diagnosis, he said, "They are possessed of the devil or bewitched." This became noised about the community, causing the children to become objects of curiosity, which naturally made them show off more. The Reverend Mr. Parris called a meeting of other ministers in the neighborhood, who, after observing, investigating and praying, unanimously confirmed the diagnosis. The supposed victims were questioned and threatened with severe discipline if they did not tell who had bewitched them, and they finally accused Tituba, Sarah Good, described as "a melancholy, distracted person," and Sarah Osburn, a bedridden old woman. The three women being formally accused were arrested and taken before Magistrates Jonathan Corwin and John Hathorne, March 1, 1692. It was arranged to hold the hearing in the village tavern (a circumstance which today will be blamed by many for all that happened afterward), but this did not prove large enough, so they adjourned to the meeting house. Sarah Good, the first to be examined, was a woman, 70 years old, in poor health and with a bad temper. All writers agree that from the first the magistrates were strongly prejudiced against the accused. The testimony also was recorded by prejudiced persons, one of whom was Mr. Parris, an accuser, and, therefore, much that was favorable was omitted from the records. Part of her examination with comments of the examiners is as follows:

Q. Sarah Good, what evil spirit have you familiarity with?
A. None.
Q. Have you made no contracts with the devil?
A. None.
Q. Why do you hurt these children?
A. I do not hurt them. I scorn it.
Q. Who then do you employ to do it?
A. Nobody.

Q. What creature do you employ then?

A. No creature, but I am falsely accused.

Q. Why did you go away muttering from Mr. Parris' house?

A. I did not mutter, but thanked him for what he gave my child.

Q. Have you no contract with the devil?

A. No.

Hathorne then desired all the children to look on her and see if this was the person who hurt them, and they all did look on her and said that this was one of the persons that did torment them. Presently, they were all tormented.

Q. Sarah Good, do you not see now what you have done; why do you not tell the truth; why do you thus torment these poor children?

A. I do not torment them.

Q. Who do you employ then?

A. I employ nobody, I scorn it.

Q. How came they thus tormented?

A. What do I know? You bring others here and now you charge me with it.

Q. Why, who was it?

A. I do not know, but it was some you brought into the meeting house with you.

Q. We brought you into the meeting house?

A. But you brought two more.

Q. Who is it then that tormented the children?

A. It was Osburn.

Q. Who do you serve?

A. I serve God.

Q. What God do you serve?

A. The God that made heaven and earth.

Comment is here made, "She was not willing to mention the word 'God.' Her answers were made in a very wicked, spiteful manner, reflecting and retorting against the authority with base and abusive words, and many lies she was taken in." Personally, I am unable to agree in this with the recorder. Samuel Abbey testified that three years previously William and Sarah Good had come to dwell in his house out of charity; that he had let them dwell there until Sarah Good became "of so turbulent a spirit, spiteful and so maliciously bent" that they could not keep her any longer. Ever since that time "Sarah Good hath carried it very spitefully and maliciously toward them." After she had gone from them they began to lose cattle, several of which were lost "in an unusual manner, in a drooping condition and yet they would eat" . . . and "doth believe that they died of witchcraft." William Good had told him that he had told his wife that they had lost two cows, and she said she did not care if the Abbeys lost all their cows. Sarah Gadge testified that Sarah Good came to her house about two and a half years previously and wanted to come in. She was told she could not because it was feared she had been with them that had smallpox, whereupon Good fell to muttering and scolding. The next morning her cows died in a sudden, terrible, strange and unusual manner so that some of the neighbors said and the deponent did believe it to be done by witchcraft.

It was here stated that Sarah's husband William had said she was either a witch or would be one very quickly. Mr. Hathorne asked him his reason for saying so of her, whether he had ever seen anything by her? He answered, "No, not in this nature," but it was her bad carriage to him. "And indeed," said he, "I may say with tears that she is an enemy to all good." From this I judge that Mr. Good had been tortured, and in this wise we still have witches.

On this evidence she was indicted, or as one writer says, she was as good as indicted before the hearing—a thing which may happen even now.

TRIAL OF THE ACCUSED

The court that tried all the cases was appointed by Governor Phips. It consisted of nine members. The chief justice was William Stoughton, the deputy governor. He had been educated for the ministry and was not a lawyer. The remaining members were citizens of prominence and influence. It is stated that while the rules of procedure were similar to those of English courts, the rights of the accused were not protected as carefully as they would have been by English judges. A prominent Boston lawyer some years ago wrote as follows: "Its proceedings were absurd and outrageous throughout; all sensible rules of evidence were ignored. There never was in any community, where the English common law was the citizens' birthright, such cruel and wanton violations of right, such absolute denials of justice as at Salem in 1692." Before they were tried the accused were treated with great severity. One writer says, "The accused were treated, from the moment some babbling child uttered a suspicious word against them to the burial of their bodies after execution, with a harshness little short of brutality, and with far more severity than any evidence would indicate that persons accused of other crimes in those days were treated." They appear to have been regarded as veritable devils themselves, ready to torment anybody. The two old women just mentioned were for several weeks taken every night to the jail at Ipswich, a distance of ten miles, and brought back in the morning. They and most of the others were kept in chains, and a bill of the Boston jailer exists which reads, "To chains for Sarah Good and Sarah Osburn fourteen shillings." It is noteworthy that one of the judges, Nathaniel Saltonstall, resigned after the first trial because "he was very much dissatisfied with the proceedings of it." The accusing persons are spoken of during the trials as "the afflicted." Their performances delayed them considerably and Thomas Newton, the prosecuting attorney, wrote to the clerk of the court, "I fear we shall not this week try all we have sent for, by reason the trials will be tedious, and the afflicted persons cannot readily give their testimony, being struck dumb and senseless for a season."

While all of the testimony is interesting reading, I have tried to detail only that which seems to be of medical and psychologic interest.

At Sarah Good's trial, Elizabeth Hubbard, one of the afflicted, said she saw the apparition of Sarah Good "biting, pricking, pinching and almost choking me to death," and that she had continued to do so until March 1, the day of her hearing. She had also seen the apparition of Sarah Good afflict Elizabeth Parris, Abigail Williams, Ann Putnam and Sarah Vibber. During the trial she was afflicted and saw the accused with invisible hands take a censer off the table and carry it outdoors.

Sarah Vibber, a woman 36 years old, said that Good tortured Mercy Lewis, April 11, and herself, May 2, by pressing her breath almost out, and also afflicted her infant so that she and Vibber could not hold it. Since then the apparition of Sarah Good had pinched, beaten, choked and pricked her with pins. Subsequently, Good's apparition came into her room one night, pulled down the clothes and looked at her four year old child, and it had a great fit. It will be noticed that this woman was not afflicted until two months after the accused was arrested. During the trial one of the witnesses cried out that Good had stabbed her and had broken the knife blade in so doing. The blade was taken from her clothes where she said she had been stabbed, whereupon a man arose who stated he had broken that very knife the previous day and thrown away the blade. He then produced the remaining part. It was shown that the girl had deliberately picked up the blade and placed it in the bosom of her dress. Sarah Good was convicted and hanged, July 19. As she stood on the scaffold the Reverend Mr. Noyes said to her, "You are a witch and you know you are a witch."

Sarah Osburn was an invalid and confined to her bed most of the time. Three people declared that she had said she was more likely to be bewitched than to be a witch. She was asked what made her say this and she replied that she was frightened one time in her sleep, and either saw or dreamed she saw a thing like an Indian, all black, which did pinch her in the neck and pulled her by the back part of her head to the door of the house. If Freud and his disciples had lived then, they could no doubt have decided her guilt or innocence by an analysis of this dream. She was sent to jail in Boston, and there died from exposure and ill treatment.

Martha Corey, a very intelligent woman, about 60 years of age, was arrested and had her first hearing March 21, 1692. It is stated that she denounced the witchcraft accusations from the beginning. Ann Putnam was her principal accuser, but she was well supported by all the others. Two of the inquisitors first paid her a visit and endeavored by cross-questioning and brow-beating to obtain a confession, but without success. One of the strongest points against her, according to them, was that on their way to visit her they had called on Ann Putnam and asked her to describe the clothes Martha had on when she made her spectral visits. Ann stated that she had just made one of these calls, but had so blinded her that she could not see what clothes she wore. When, however, they were questioning the accused, she inquired "if the afflicted had attempted to describe her clothes." This they claimed showed supernatural wisdom on her part. As a matter of fact, it was a proper question as in all previous cases the clothing worn when they made their visits had been described. They also made much of her statement that "she did not believe there were any witches." A sample of the examination at her first hearing is:

Hathorne. You are now in the hands of authority. Tell me now why do you hurt these persons?

A. I do not.

Hathorne. Who doth?

A. Pray give me leave to go to prayer.

Hathorne. We do not send for you to go to prayer, but tell me why you hurt these.

A. I am an innocent person. I never had to do with witchcraft since I was born. I am a gospel woman.

Hathorne. How could you tell then that the child was bid to observe what clothes you wore when some one came to speak with you? (Cheever interrupted and bade her not begin with a lie.)

Hathorne. Who told you that?

A. He said the child said—

Cheever. You speak falsely.

Hathorne. Why did you ask if the child asked what clothes you wore?
A. My husband told me the others told.

Hathorne. Goodman Corey, did you tell her?
The old man denied that he had.

Hathorne. Did you not say that your husband told you?
No answer.

Hathorne. How dare you lie to this assembly? You are now before authority. I expect the truth. You promised it. Speak now, and tell who told you what clothes.

A. Nobody.

During this one of the children exclaimed that a man was whispering in Martha's ear. She was asked, "what did he say to you?" Her reply was, "We must not believe all that these distracted children say."

The Reverend Mr. Parris in his report writes: "It was noted when she bit her lip, several of the afflicted were bitten. When her hands were at liberty the afflicted were pinched." It was asked, "Do you not see that these women and children are rational and sober when your hands are fastened?" Immediately they were seized with fits and the bystanders said she was squeezing her fingers. Some one exclaimed, "She hath bit her lip, and immediately the afflicted were in an uproar." During this examination she was badgered by every one present, especially by the Reverend Mr. Noyes. A Mrs. Pope threw her muff at her and followed it with her shoe which struck her on the head. This woman was an important witness in several cases and subsequently acknowledged her error. Mrs. Corey was hanged, September 22, previous to which she was excommunicated by the Reverend Parris and two deacons of the church. Shortly after Giles Corey, her hus-hand, 80 years old, was arrested. He had testified against his wife. During his examination the afflicted suffered so much from fits and pinches that his hands had to be tied. When he turned his head to one side, their heads were likewise turned, and when he drew in his cheeks, their cheeks were sucked in. What was considered strong evidence was given by a 16-year-old boy, who said that about August 20, Giles Corey told him that he wanted some platters as he was going to have a feast. . . . He took them and carried them away, being gone about a half hour, then he brought them back, went away and said nothing. It was believed that it was not Giles Corey in the flesh who had either borrowed or returned them. At his trial in September he refused to plead, or "stood mute," as the law termed it. For this he was pressed to death. This consisted in laying the victim naked on his back on a hard floor and placing on his body "as great a weight of iron as he could bear and more." He was given on the first day three morsels of the worst bread, and on the second day three draughts of standing water that should be near the prison door, and in this situation this should be alternately his daily diet until he either answered or died. He lived two days. This is said to be the only case in which this law was enforced in New England.

Rebecca Nurse was the mother of eight children and in good circumstances. She was accused of sundry acts of witchcraft directed toward Mrs. Ann Putnam, Ann Putnam, Jr., and Abigail Williams. Her formal accusers were Thomas and Edward Putnam. During the hearing Mrs. Ann Putnam cried out, "Did you not bring the black man with you? Did you not bid me tempt God and die? How often have you eat and drunk your own damnation?" To this the accused replied, "O Lord help me," and spread out her hands, whereupon it is recorded that "the afflicted were grievously vexed." Her answers to all questions were particularly straightforward and sensible. When asked if she thought the afflicted suffered against their wills or not, she replied that she did not think they suffered against their wills, and she did not believe they were bewitched. She was asked why she had not visited the afflicted. She answered that she was afraid she would have fits also, whereupon all the afflicted had fits abundantly. Every time she moved her hands the afflicted persons were seized with violent fits of torture. If she held her head to one side the afflicted also held theirs, and one of them had her neck set in that position. Seeing this, another cried out, "Set up Goody Nurse's head, the maid's neck will be broken." When some one set up Nurse's head it was observed that the afflicted one's was immediately righted.

At her trial it was claimed that she had a "witch mark" on her body. A jury of women was appointed to examine her. They disagreed, some very properly claiming that the mark was due to natural causes. The prisoner herself said that she could give "a sufficient known reason of the moving cause thereof," and asked to be examined by another jury, which request was apparently not granted. Two women also sent a statement to the court as follows: "We whose names are underwritten can testify if called to it, that Goody Nurse has been troubled with an infirmity of body for many years, which the juries of women seem to be afraid it should be something else."

The following written statement sent to the court by John Tarbell is of interest:

> John Tarbell being at the house of Thomas Putnam upon the 28th day of March, 1692, upon discourse of many things, I asked whether the girl that was afflicted did first speak of Goody Nurse, before others mentioned her to her. They said she told them she saw the apparition of a pale faced woman, that sat in her grandmother's seat but did not know her name. Then I replied and said, but who was it that told her that it was Goody Nurse? Mercy Lewis said it was Goody Putnam that said it was Goody Nurse; Goody Putnam said it was Mercy Lewis that told her; thus they turned it upon one another, saying it was you and it was you that told her. This was before any was afflicted at Thomas Putnam's house besides his daughter and that they told his daughter it was Goody Nurse.

A verdict of not guilty was rendered, whereupon all the accusers in court cried out with renewed vigor, and were seized with violent fits, rolling and tumbling about, creating a scene of the wildest confusion. The judges told the jury that they had not carefully considered one expression of the prisoner, i. e., that when a confessing witch was brought as witness against her she said, "What do you bring her? She is one of us." Even then they could not agree on a verdict of guilty, but returned to the courtroom and asked the prisoner to explain the remark. She made no reply, whereupon they found her guilty. It later appeared that she was deaf and had not heard the question. When she was made to understand it she stated that she had meant the witness was a fellow prisoner. This explanation, a perfectly good one, was never told the jury. Her conviction was forced by the court which should have protected her interests. She was sentenced to be hanged, but was reprieved by the governor. The church thereupon excommunicated her, and the afflicted renewed their clamor and symptoms. This, together with the influence of some Salem people, caused him to withdraw the reprieve and she was duly hanged. It is noteworthy that the Rev. George Burroughs, who was accused and executed, had had a financial dispute with the Putnams, who were active as his accusers, and their daughter Ann was the most important witness against him.

At the trial of Bridget Bishop, the Rev. John Hale testified that the wife of John Trask desired of him that Bridget Bishop be not permitted to receive the Lord's Supper because "she did entertain certain people in her house at unseasonable hours in the night to keep drinking and playing at shovel board whereby discord did arise in the other families and young people were in danger of being corrupted." He greatly feared that if a stop had not been put to these disorders Edward Bishop's house would have been one of great profaneness and iniquity. It was later said that on the night Mrs. Trask made this complaint she became distracted and remained so until she died. Other records show that she was insane and committed suicide by cutting her throat. George Jacobs, Sr., a well-to-do man of 70 years, was accused by his servant, Sarah Churchill, one of the original accusers. The principal evidence against him was his inability to repeat the Lord's Prayer. His son George Jacobs, Jr., and his wife and daughter were shortly after arrested. The son escaped, but his wife was kept in irons for eight months and then acquitted. The daughter to save herself, confessed that she was a witch, and testified against her grandfather who was hanged. She afterward confessed that she had been made to do this. Part of the confession is as follows:

> The Lord above knows I know nothing in the least measure how or who afflicted them, they told me without doubt I did, or else they would not fall down at me. They told me if I would not confess I should be put down into the dungeon and would be hanged, but if I would confess I should have my life. The which did so affright me with my own vile, wicked heart, to save my life made me make the like confession I did. Which confession may it please the honored court, is altogether false and untrue.

At the trial of Elizabeth Procter the court addressed the affected ones as follows:

> Mary Walcott, doth this woman hurt you? Ans. I never saw her so as to be hurt.
>
> Mercy Lewis, doth she hurt you? (Her mouth was stopped.)
>
> Ann Putnam, doth she hurt you? (She could not speak.)
>
> Abigail Williams, doth she hurt you? (Her hand was thrust in her own mouth.)

Later, Abigail Williams and Ann Putnam were again spoken to by the court, but neither of them could make any answer by reason of dumbness or other fits. By and by they cried out, "Look you there is Goody Procter on the beam," which statement was followed by the accusing of John Procter, the husband, who had come with his wife to court. At their trial the following evidence was given by Elizabeth Booth:

> On the 8th of June Hugh Jones appeared to me and told me that Elizabeth Procter killed him because he had had a pot of cider of her which he had not paid for. On June 8th Elizabeth Shaw appeared unto me that Elizabeth Procter and John Williams killed her because she did not use those doctors she advised her to. . . . Ye wife of John Fuller appeared unto me and told me that Elizabeth Procter killed her because she would not give her apples when she sent for some. . . . The apparitions of Law Shapling and Doc Zerubabel Endicott said Elizabeth Proctor killed them, and the apparition of Robert Stone, Senior, told her that John Procter and his wife killed him, and at the same time Robert Stone, Jr., appeared and said Procter and his wife killed him because he took his father's part.

John Bailey testified to seeing the Procters standing at the door of their house (at which time they were in jail), and after getting a mile from it he became speechless for a time, also that after alighting from his horse he saw a woman coming toward him, but that when he got on his horse again "there stood a cow where I saw the woman." As evidence against John Procter it was stated that at the beginning of the witchcraft prosecutions he had said in reference to the afflicted that he could whip the devil out of them, which seems to be a very reasonable statement, and probably would have caused the witches to vanish. A man stated that he had heard one of the accusing girls say that she had cried out against Goodman Procter for sport. "The girls must have some sport," she added. He testified in his defense that five persons, one of them his son, had been tied neck and heels until the blood came from their noses, in an effort to have them confess. John Procter was hanged; Elizabeth was condemned, but not executed. A family by the name of Wilkins having considerable illness sent for Mercy Lewis and Mary Walcott (heretofore active as accusers) to ascertain the cause. They promptly saw the apparitions of Sarah Buckley and John Willard on the throat and breast of Henry Wilkins. Willard was promptly arrested. At his trial, Ann Putnam testified that the shapes of Samuel Fuller and Lydia Wilkins had appeared in winding sheets and told her, "If I did not go and tell Mr. Hathorne (a magistrate) that John Willard had murdered them they would tear me to pieces." He was hanged.

Martha Carrier, a woman 40 years old, was arrested with four of her children. During her trial "the tortures of the afflicted were so great, that there was no enduring it, so that she was ordered away to be bound hand and foot, the afflicted in the meantime almost killed. As soon as she was bound they all had strange and sudden cease." Her daughter, 8 years old, testified against her, saying she had been a witch since the age of 6 and her mother made her so. Her son, aged 18, also testified that he had been "in the devil's snare," and that one of his brothers was also. A neighbor stated that after some dispute with her, he had been taken with a swelling in his foot and subsequently had two sores in the groin. The swelling was lanced, and a large amount of corruption came away. The very day that Goodwife Carrier was arrested, he began to get better. Therefore "he had great cause to think that she had a hand in his sickness." She was hanged.

The first charge against Elizabeth How, said to be a kind and charitable woman, was made by a girl 10 years old, whose parents had previously had a dispute with the How family concerning some lumber. At her trial, it was stated this girl had told reputable people "that if she did complain of Elizabeth How in her fits she did not know that she did so," and that they had heard her brother tell her to say that Goodwife How was a witch. In spite of this she was hanged.

At the trial of Susanna Martin, one of her neighbors said that her reason for believing her a witch was her great neatness. For this she was hanged.

Mary Easty was released after trial. Two days after her discharge, Mercy Lewis had a fit. Ann Putnam was sent for to tell who afflicted Mercy. She and Abigail Williams visited her and declared they saw Mary Easty and John Willard (before mentioned) afflicting her body. She was rearrested at midnight and taken to jail, loaded with chains and finally executed.

Abigail Hobbs, who apparently was not well balanced, declared that while in jail Burroughs (later executed) come to her "in his body person," bringing of a half dozen girls for her to afflict by sticking thorns into them, and when she pricked them the real girls cried out with pain and she heard them. She also felt the hand of Burroughs on her arm. In conclusion, she stated that she had killed both boys and girls. She also accused her mother and father. They denied the charge, but after repeated questioning the mother finally confessed that she was a witch and that her husband was also.

Mary Warren, who had been one of the accusers of John Procter, before mentioned, and had testified in other early cases, later said that the afflicted "did but dissemble." The other girls then cried out against her and she was arrested. Part of the record of her examination is as follows:

> After continuing in a fit for some time she said, "I will speak, O, I am sorry for it, I am sorry for it." Wringing her hands she fell into another fit. Then attempting a little later to speak, her teeth were set. She fell

> into another fit and shouted, "O, Lord help me, O, good Lord save me," then afterward cried again, "I will tell, I will tell," and then fell into a dead fit again. . . . She said, "I must not speak a word, but I will speak, I will speak, Satan. . . . Avoid Satan, for the name of God, avoid." She then fell into fits again.

She was later released and resumed her occupation of testifying.

Rebecca Eames, while a spectator at some of the executions, was accused by a woman of sticking a pin into her foot and of bewitching her. She was arrested and later confessed that she was a witch. She was sentenced, but not executed. In October, 1692, a new court was appointed which was even more in sympathy with the prosecutions than the preceding one had been. That there might be no question as to their right to hang witches, they had the Colonial statute against witchcraft reenacted, and a few months later reenforced it with the English statute. In spite of this they could not get juries to convict. Two reasons have been given for this; one, because the wife of a clergyman previously active in the prosecutions was accused, the other because "the juries who tried cases in 1692 were composed of freemen only, while those of 1693 were chosen from among all those inhabitants who possessed the requisite amount of property to qualify them as electors under the new charter. Freemen were necessarily church members and not as likely to act independently as the jurors selected from the whole body of the people." Protests began to be made by various communities, and the last three convicted and sentenced were not executed. In May, 1693, Governor Phips issued a proclamation releasing from custody all persons, about 150, held on the charge.

THE PSYCHOLOGY OF THE OUTBREAK

A number of writers have discussed the psychology of this outbreak, notably, Lecky, Munsterberg, Beard and Kittredge. To me, it seems comparatively simple. It was due to hysteria, fomented by religious fanaticism and lying, some possibly of the pathologic type, but most of it malicious. It must be borne in mind that the Puritans were a fanatical and bigoted people, who persecuted vigorously all those who did not agree with their religious convictions. In spite of the fact that they themselves had come to this country to escape persecution on account of their religion, in Salem Quaker women had been dragged half naked through the streets at the tail of a cart and whipped. Baron Pollock, an English lawyer wrote, "It is probable that but for the persecution of heretics, there would have been no persecution of sorcerers." These people led a more or less repressed life and were therefore ripe for any orgy when the opportunity came.

One writer has suggested that much of the evidence was due to hypnotism. In only one instance does this seem likely, i. e., the young girl who testified against Elizabeth How, said she did not remember making the charge. All the others did remember everything they said and did.

A number of symptoms of hysteria were present. The influence of suggestion and the morbid desire for notoriety are apparent. In each succeeding trial the symptoms became more pronounced and bizarre. Mimicry was frequent, and in one case possibly a hysterical contracture was present. Some had areas of anesthesia and hyperesthesia. These symptoms were encouraged maliciously by older people. It is noteworthy that many of the accused had had difficulties, either legal or otherwise, with their neighbors, or were more or less public nuisances. Also many confessed to being witches and testified against the accused because it was known that those who did so would not be executed. It is also evident that in some cases torture was used to get confessions. Hutchinson, in his history of Massachusetts, writes, "The whole was a scene of fraud and imposture, begun by young girls who at first perhaps thought of nothing more than being pitied and indulged, and continued by adult persons who were afraid of being accused themselves." Many of the active instigators of these prosecutions afterward acknowledged their error, and expressed regret. This did not, however, help those who had suffered.

MODERN WITCH CRAZES

I suppose most people will say that such a thing could not occur today. I believe that it is not only possible, but to a certain extent is already occurring. We are doing many things similar to what these people did. George M. Beard, in his book, "The Psychology of the Salem Witchcraft Excitement in 1692," writes, "that he who should on a wager contract to excite the American people through the emotions, and in that way convert them to any doctrine within three months, and to a denial of the same doctrine within another three months, could if he were moderately skilful, gain his contract and win his wager." This was written about forty years ago, but it is more true today. The people of Salem and its vicinity were justified in believing in witchcraft. Its existence was generally taught by clergymen and others whose opinions were entitled to respect. Where they erred was in depriving the accused of their legal rights, in treating them with unusual severity and in refusing to consider evidence in their favor. Cotton Mather who, as is well known, was one of the most influential men in the colony, wrote to the judges to exercise great care and not to convict on spectral evidence alone. He further stated, "It is certain that devils have sometimes represented the shapes of persons not only innocent, but very virtuous." No attention was paid to this, and convictions were repeatedly secured on this evidence, which was contrary to all the rules of evidence then maintaining. The uproar at the trial of Rebecca Nurse puts one in mind of the newspaper accounts some months ago of the hearing on the question of Sunday music in this city before a committee of the legislature. It is said that speakers in favor of the measure were hissed, villified and their voices drowned in the uproar made by the opponents, largely composed of clergymen. In spite of the facts that concerts on Sunday evening are held in many American cities without evident damage to the population and that certain religious denominations are not opposed to them, statements were made of which this is a sample: "People who recently have come to these shores from lands overrun by bolshevism are the principal opponents to the blue laws." Is this true? When the park commission allowed the playing of baseball in the park on Sunday a well known clergyman at a public meeting characterized it as "anarchy and bolshevism of the worst kind," also that "the park commission have not only broken the Sabbath day, but have committed a very serious offense to our nation, to its laws and the great democracy that endeavors to set an example before the world." When he said this he probably knew that in many places in our nation baseball is played on Sunday, not only for pleasure, but also for gain. We are now suffering from a surfeit of legislation and proposed legislation regulating our habits, our business, in fact, practically everything good or bad that a person is liable to do. To quote a recent editorial:

> Getting a legislature to act upon all manner of essentially personal and petty questions is perfectly easy. This state solemnly forbids playing any game of cards in a public resort. That one forbids buying a cigar on Sunday. A number have gravely dealt with the portentious question of giving tips. Every winter produces a sheaf of bills to regulate women's dress. Getting any state to attend to such matters as how and when a kimono shall be worn, or the length and cut of bathing suits, is perfectly easy. . . . But getting a legislature ever to consider public questions of large importance is difficult. . . . Where the law requires a washerwoman's cook stove to be assessed, and a millionaire's wife's diamonds are not assessed, the legislature is deeply engaged with the censorship of moving pictures. Where mob murders are a well recognized institution, the legislature is passing an act to regulate the length of hat pins.

A special target for suppression by legislative enactment seems to be anything which may add to the pleasure and relaxation of the individual. Not long ago, a clergyman publicly stated that moving pictures are one of the biggest menaces leading to vice in this country. To this end we have numbers of societies whose mission is to regulate some one particular thing and that are the instigators of much of this sort of legislation. Not only this, but there are societies whose mission is to regulate public and private institutions, private business, education; in fact, nothing escapes. The prevailing motto seems to be, Attend to everybody's business but your own, and the less you know about the business you are regulating, the better. To quote Henry Watterson:

> Under the pretense of liberalizing the government, politicians are sacrificing its organic character to whimsical experimentation, its checks and balances, wisely designed to promote and protect liberty, are being loosened by schemes of reform more or less visionary; while nowhere do we find intelligence enlightened by experience, and conviction supported by self control, interposing to save the representative system of the Constitution from the onward march of the proletariat. . . . When they have not gone skylarking or have grown tired of bridge they devote their leisure to organizing clubs other than those of the uplift. There are all sorts, from the Society for the Abrogation of Bathing Suits at the seaside resorts to the league over at Mewville for the care of disabled cats. Most of these clubs are all officers and no privates. That is what many of them are made for. Do they advance the world in grace? One who surveys the scene can scarcely think so.

While many of these organizations are public nuisances, others when properly conducted do much good. Their usefulness is often impaired by lack of tact and respect for the rights and feelings of those to be benefited. This is often due to the lack of knowledge of the world and its people possessed by those who carry on the work. It may be illustrated by a story said to be true:

> A wealthy lady entertained some poor children in her neighborhood. During the afternoon one of them asked her if her husband drank, to which she replied, "He takes a drink now and then but never to excess." Then the child asked, "Does he ever beat you?" The lady, becoming very indignant, exclaimed, "Child, how dare you ask me such a question?" Whereupon the child answered, "My mother told me before I came here to act like a perfect lady, and that is what I heard you ask her."

The feelings of many of these objects of solicitude seem to be well described in the following verses appearing in a daily paper not long ago:

Oh, a workman who was weeping with despair,
Cried, "The women's clubs have tracked me to my lair.
I will flee the whole caboodle
In this land of Yankee Doodle
Where there's nothing but compulsion everywhere.

"They will legislate the pattern of my shoes.
They will censor my amusements and my news.
I can have no more endurance
With their plans for new insurance
Which will eat my little income up with dues.

"Oh! I am weary of the struggle and the strife
Gainst this most efficient legislated life.
For my uplift they are burning,
While I am all their efforts spurning,
Ere they make me wed a legislated wife."

Many of these organizations and individuals who are ambitious to have the world run according to their ideas, in order to gain their ends are guilty of false and reckless statements, of advocating the confiscation of legally owned property, of breaking laws themselves in order to capture those who in their opinion are breaking laws, and of villifying and slandering those opposed to them. We have heard that those who prosecuted the witches in Salem were guilty of many of these things, and it seems probable that with a little more power the modern regulators will advocate the electric chair for those who differ from them. A still more serious thing is that those whose duty it is to enforce the law engage in orgies of persecution in which people's rights are trampled on with impunity and much injustice is done, frequently without redress. Russia in the days of the czar had something to learn. We are going on the theory if one person may be made good (who does not want to be) it is proper to make thousands miserable. To emphasize these statements, let me abstract some of the news appearing in the daily papers of the past few years. They are not isolated cases, a book could easily be filled with similar ones. July 26, 1918, a public, licensed hotel in the center of the city was raided on the ground that it was a resort for immoral people. At the time, the officers smashed down doors, ripped out transoms and broke open trunks in the search for evidence. They arrested forty or fifty men and women, not only those who were in the place, but those who were on the street in the vicinity. When tried the first time, the proprietor was convicted, but the judge promptly gave him a new trial, and stated:

> No man had the right or justification to break into another man's house, commit depredation, or rummage through his personal belongings without a warrant, even if he knows he will find incriminating evidence. Any man who breaks into another man's house without a warrant and accuses that man of violation of the law, in the same breath admits he has violated the law himself.

At the next trial he was acquitted. The trial judge (not the one who conducted the first trial) in his charge said:

> This raid was conducted in a high handed and illegal manner. . . . These agents took it upon themselves to disregard the law and the rights of citizens, to engage in an unlawful and unwarranted act.

Such performances are supported by people supposed to be law abiding. One may ask what will happen when they secure judges who will do their bidding.

About the same time, three women, each about 60 years old, were arrested for frequenting an immoral resort (their own home). The only evidence was that one of them had three sons in the Navy. A man, enlisted in the Navy, at the instigation of the federal law enforcing officer for the suppression of vice, telephoned her, claiming to be a shipmate of the sons, and asking leave to call. This he did with a companion. After two of the women had gone to their rooms to dress, the men forced themselves into their rooms. Others who were waiting outside then forced themselves into the house and arrested the women. They were acquitted, the trial judge characterizing the case as one of the most outrageous he had ever heard of.

In Newark, N. J., not long ago, policewomen were ordered to forcibly wash the faces of any girls on the street with painted cheeks, and to have them photographed as vampires.

In New York, in 1918, a wave of hysterical morality caused the raiding of public hotels, apartment houses, restaurants and billiard rooms, with the arresting, without warrants, of 1,100 people. All this to find possible hidden vice. Shortly after Rodman Wanamaker, special deputy police commissioner, advised the use of women to act as police spies in the following way:

> Women of character and ability can be easily found to volunteer for this work. . . . It can be extended . . . so as to have in every block of inhabited territory in this city a woman in charge, whose post it would be and whose duty it should be to keep that block clean and wholesome by becoming acquainted with every person and condition existing therein and reporting as may be directed by the police commissioner everything unlawful or irregular coming to her notice.

A delicate tribute to a well-known characteristic of many women. According to the papers the services of the emergency aides were offered in this city for such work.

About two years ago, a so-called vice crusade was instigated in Wilkesbarre by the chamber of commerce there. It was afterward stated by one of the detectives employed for the work that the chamber of commerce had supplied him with $10,000 to be used in luring innocent girls to houses of ill-fame and otherwise to test the vice conditions of the city.

Recently a prominent aviator of the United States Army, Lieutenant Maynard, was quoted by the Anti-Saloon League as saying that a large number of the casualties which occurred in the recent transcontinental race were caused by flying with hangovers or when drunk. This was published far and wide by the newspapers and a sensation made of it. A frequent heading was "Booze Knocked out Transcontinental Fliers." This was absolutely denied by the gentleman in question. He said that his real statement was, "Many accidents in aviation are caused by the use of intoxicants," and that the Anti-Saloon League had deliberately juggled this to suit their own purposes.

Many of the active advocates of prohibition advocated practical confiscation of the property of those engaged in the manufacture and sale of alcoholic drinks, and also that of private owners. Whatever one may think of the merits or demerits of prohibition, sane people must recognize that those engaged in a previously legal and exceedingly old business should be dealt with fairly. In this country during the war tavern keepers who had paid a license and whose capital was invested in their business were compelled to close without compensation, when located within certain prescribed areas. England when she did this under like conditions compensated all such.

Certain organizations, which after spending large sums of money to make this country dry, are now, according to the papers, raising large sums to put the world in a similar fix. Yet when a well known newspaper man borrowed money from some brewers in order to purchase a newspaper it was made the subject of a congressional investigation.

In Virginia, as part of the operation of the search and seizure act of that state, we find that police opened the coffin containing the body of a well known person that was being transported by railroad through the state. Women in sleeping cars had their berths searched by male officers while occupied by them, and a number of suspects were shot and killed.

Tobacco is at present in serious danger of execution for witchcraft. Interesting, if not true, are the statements of the Rev. Clarence True Wilson (there should be a question mark after the middle name), who is secretary of the temperance board of the Methodist church: "The cigarette impaired the health of two million soldiers of our Army. We have been sold out by the White House band to the tobacco trust. . . . The American Tobacco Company sent to the soldiers in France doped cigarettes which succeeded in making many of them virtual dope fiends, unable to fight for two hours without collapsing unless they had their smokes." Cigarets had to be pushed into the mouths of the trembling soldiers before they could go over the top, and ignorant doctors called this shell shock. So far as known, these statements have not been denied by newspapers or medical journals.

In a number of states, the sale, and even the smoking, of cigarets is unlawful. In Kansas, newspapers and magazines advertising them cannot be publicly sold. In this same state the superintendent of public instruction (a woman) has forbade the employment of male teachers who use them. Fortunately for education in Kansas, she did not say anything about the female teachers.

It is of interest to note that in Nebraska, for some time a very dry state, where cigarets are prohibited, and where a bill was proposed in the legislature making it a penal offense to have the odor of an alcoholic drink on the breath, not long ago the governor was nearly murdered and the court house burned in Omaha by a mob who wished to and eventually did lynch a negro accused of a certain crime, but not yet proved guilty. Also in Oklahoma with similar laws, a United States senator was driven from the stage with eggs in one of its towns while attempting to make a speech against the League of Nations.

It will be found by investigation that most lynchings and other outbreaks of mob violence occur in states most prone to freak legislation. This seems to show the lack of mental balance of the people. A member of Congress is recently quoted as stating that there were 80,000 drug addicts in the first draft, and that they were all rejected. The second report of the Provost-Marshall General says that of 3,208,446 men examined, 2,007 were rejected for drugs and alcohol — quite a difference.

In a Missouri mining town a few years ago, 700 foreign born laborers were driven away from their homes by American born miners armed with shotguns, who insisted that all foreign born miners must leave.

During the late war our hysterical temperament was strongly manifested; and when started our antipathy for anything German was much stronger and is now much stronger than in those countries that suffered most from the war. It may be remembered that during the summer of 1918, when it was requested not to use automobiles on Sundays, a gentleman in a nearby town took his wife, who was a cripple, to church in his motor car. While he was in church, a mob cut his tires, emptied his gasoline tanks, damaged his car and subjected him to verbal abuse. The newspapers were full of accounts of physical and other abuse inflicted on those who did not subscribe to liberty bonds. In some cases possibly there was reason for this; in others there was not. In a town in Nebraska, a hall which had been used before the war as a meeting place for German societies was burned during the war by incendiaries, and the fire department made no effort to save it.

One of the results of this peculiar mental state, which I have tried to show now afflicts many of the people of this country, is the meddling with the management of institutions, such as prisons and hospitals for the insane, especially the former. The officials of these institutions are very much in the position of the Salem witches, and are subjected to all sorts of accusation and abuse, usually by people who have no practical knowledge of the subject at all, sometimes by those who have, but who, courting notoriety, want to be right with the crowd. The newspapers, without knowledge of the facts, take great delight in making a sensation of the most trivial charges.

What joy the sob sisters must have received from this account published in a daily paper, concerning a gentleman (we must call them so now-a-days) who was in the highway robbery business (now very popular) and departed this life by suicide in a penal institution not long since. "Billy loved life too much ever to attempt suicide," said the youngest sister as she sobbed on the shoulder of her older sister. "No one can tell us he killed himself. He had too much 'pep,' and too much to live for."

No doubt there are still many people who need to be separated from their valuables by use of the sandbag and other gentle means of persuasion.

A PLEA FOR SANITY

Possibly some may call me an iconoclast. I am not. I believe heartily in all measures that help the poor and unfortunate, provided, as I have already said, the advocates of these measures and those who carry them out, act with decency and common sense, and not as hysterics and notoriety seekers. Social service, for instance, is a grand thing when it is not secret police service, as for example, when some years ago I was requested, almost ordered, to send a widow, aged 40 years, to the hospital for the insane because she drank beer with men. Needless to say I refused. The same may be said of a number of other organizations and measures. They are useful when sane.

I have tried to show that much is done now in the name of law, order and uplift that is not sane. Dr. Lloyd, at a meeting of this society a few months ago said: "The various crazes of today are an evidence of the unsettled minds of the community." They are therefore legitimate subjects for the attention of this society. Not a little of freak legislation is proposed by medical men, which makes it doubly important that we as medical men and women who study especially mental diseases, endeavor to guide such minds, both medical and lay, into proper channels.

To my mind these are serious days, and anything which promotes morbid and hysterical mental states is dangerous, and worthy of our earnest consideration. Otherwise, we may have a witch craze on our hands that will make that of Salem appear very small, indeed. To quote again Henry Watterson:

> Bigotry is a disease. The bigot pursuing his narrow round is like the bedridden possessed by his disordered fancy. It sees nothing but itself, which it mistakes for wisdom and virtue. But it also begets hypocrisy. When this spreads over a sufficient area and counts a voting majority it sends its agents abroad, and thus we acquire canting apostles and legislators at once corrupt and despotic. . . . The worst of it is that there seems nowhere any popular realization—scarcely any popular outcry. . . . In a nation of undiscriminating voters the voice of the agitator is apt to drown the voice of the stateman.

LETHARGIC ENCEPHALITIS

A REPORT OF FOUR CASES AND AN ANALYSIS OF ONE HUNDRED CASES REPORTED IN THE LITERATURE *

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It seems worth while to report the following cases observed in the Waterbury Hospital, together with a review of the literature and an analysis of one hundred cases regarded by us as typical.

REPORT OF CASES

CASE 1.—*History.*—Miss F. S., aged 25, first complained of diplopia on Jan. 28, 1919. Although at first this was transient, it became more persistent and disturbing. About the same time her mother noticed that the patient, her daughter, had a slight drooping of the right upper eyelid. Except for these symptoms the history was negative. February 5, when the patient complained of headache and general malaise, she was drowsy and answered questions with apparent reluctance. The patient was admitted to the Waterbury Hospital on February 8, with a temperature of 100; pulse, 96, and respiration, 20.

Examination.—She had a dull facial expression. There was drowsiness with marked reluctance to answer questions, although on insisting, they were answered quite intelligently. There was external strabismus of the right eye with ptosis; the pupils were equal and reacted to light; the accommodation reflex was hard to obtain because the patient did not focus on the object. Whistling, wrinkling of the forehead and grinning showed nothing abnormal. The tongue was coated and protruded in the median line; there was no tremor. Heart and lungs were normal. The bladder was distended and 28 ounces of urine were obtained by catheter. There was no rigidity of the neck, the knee jerks were slightly exaggerated, Kernig, Babinski and Brudzinski signs were absent.

Hemoglobin, 90 per cent.; red blood cells, 5,100,000; white blood cells, 7,400; polymorphonuclear cells, 76; lymphocytes, 21; mononuclear and transitional cells, 3; eosinophils, 0 per cent. The urine showed specific gravity 1.026; there were no albumin, no sugar and no casts; there was a very slight trace of acetone and no indican. The blood pressure was systolic 110, diastolic 78.

A lumbar puncture was performed on February 14. The fluid was not under pressure, was clear with 10 cells per c.mm., and there was a very slight increase of globulin; Fehling's was slightly reduced. No organisms were found either by smears or by cultures; the antiformin method was used, but tubercle bacilli were not found. The Wassermann was negative both in the blood and spinal fluid.

Course and Treatment.—During the first week in the hospital her condition remained about the same She was lethargic, lay on her back unless turned, did not speak unless an answer was insisted on, did not ask for food. She had to be fed with a spoon, but there was no dysphagia. The ptosis was constant, but the strabismus varied in extent. The temperature varied from 98 to 100 F.; pulse from 78 to 110; respiration from 17 to 24.

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* From the Medical Service of the Waterbury Hospital.

At the end of the first week the drowsiness became less and the strabismus inconstant. Irregular jerkings, a very marked and coarse intention tremor, and apparent incoordination of both upper extremities became evident. Eye grounds were normal on the day of admission and also seven days later.

A blood count was made every other day (Table 1). The systolic blood pressure varied from 108 to 118; the diastolic from 70 to 76.

TABLE 1.—Total and Differential Leukocyte Counts (Case 1)

Date	White Blood Cells	Polymorphonuclears	Lymphocytes	Mononuclears	Transitionals	Eosinophils
Feb. 8	7,400	76	22	2	0	0
Feb. 10	6,800	72	17	1	2	1
Feb. 12	8,200	79	17	1	0	1
Feb. 14	7,800	74	19	1	0	1
Feb. 16	9,200	76	18	2	2	2
Feb. 18	6,800	70	16	1	2	1

The results of creatinin and sugar determinations made of the blood were: creatinin 1.1 mg. and sugar 45 mg. per 100 c.c. of blood.

During the second week the patient's condition improved rapidly. The retention of urine entirely disappeared and the bowels moved after simple laxatives. She gradually became more alert and by the end of the second week was mentally normal. When asked to describe her feelings and her mental state of the week before, she said that she could not, as she remembered only that she had been brought to the hospital and was catheterized several times. The patient was discharged as cured on March 4.

Case 2.—*History.*—M. S., female, aged 6, entered the hospital, July 14, 1919. The family history was negative. She had had smallpox several years before and influenza in October, 1918. Her present illness began three weeks before admission and was ushered in by vomiting several times during twenty-four hours. On the next day she complained of severe abdominal pain and was put to bed, where she remained for seven days. During this time the abdominal pain was quite distressing and the vomiting frequent and persistent. During the second week she felt better and began to walk about, but her parents noticed that her actions were peculiar and that she spoke unusually loudly. During the latter part of the week squinting of the left eye was noted; also partial bilateral ptosis which was essentially marked at night. In the third week she was again put to bed because of abdominal pain and vomiting. The pain soon subsided. The left external strabismus persisted, but the right eyeball lost all movements. Her intelligence at this time was good, but she spoke very little and had lost the loud tone of voice. Dysphagia and inability to walk were first noticed at this time.

Examination.—On admission to the hospital she was well nourished, very drowsy, with a temperature of 100.2; pulse, 100; respirations, 22. There was external strabismus of the left eye with slight facial palsy and ptosis on the same side. There was no rigidity of the neck, but a suggestion of Kernig's sign. Heart and lungs were normal. Deep reflexes were slightly exaggerated; the Babinski sign was negative. There was incontinence of urine and feces. She could be aroused to answer simple questions, but none that required much thought; she could differentiate large from small objects and could distinguish one coin from another. Soon after answering questions she would again sink into lethargy, occasionally uttering little whines, but for the most part remaining quiet and apparently asleep. She would awaken for meals, but resisted feeding because of dysphagia. This necessitated gavage. After several weeks in the hospital she became stuporous and more restless. The right arm and hand moved incessantly, trying to grasp one's hand when approached. The left extremities appeared to be partially paralyzed. Her eyes stared inattentively. At this time there was right internal strabismus; the pupils reacted moderately to light but were irregular, the right being more dilated. Eye grounds were normal during the entire illness.

Course and Treatment.—About August 1 the patient seemed to be in deep coma. The right arm and leg were very inactive, but responded to painful stimuli. Sensation and acuteness of the special senses were much lessened. Corneal reflexes were much diminished, and "films" persistently formed over the corneas. In the parts exposed by the open eyelids there was also a low grade conjunctivitis which required constant attention.

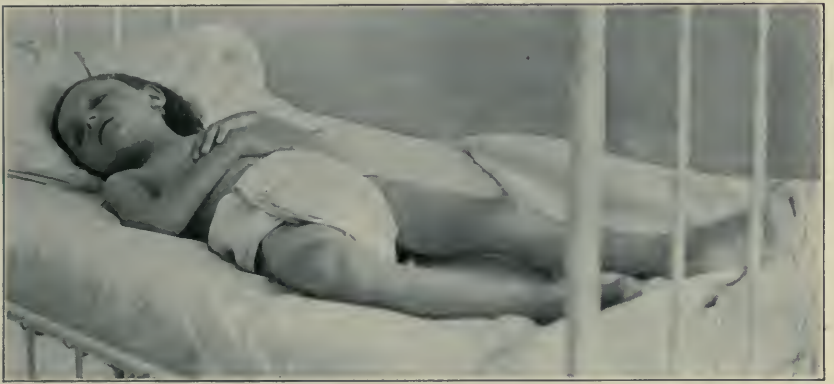

Fig. 1 (Case 2).—Appearance of patient five days after admission to hospital.

During this time the patient would not swallow any food or medicament, the most careful attempt leading to alarming spells of choking and coughing. She was fed by tube for five weeks; food with a value of from 1,000 to 1,200 calories was introduced daily, which kept weight and color good.

The heart action was rapid but fairly strong. At times, however, the pulse rate would rise to 150 or 160, and would be irregular in force and rhythm. With this acceleration of pulse rate there was irregular breathing, profuse perspiration and choking. These attacks occurred mostly at night. Records of the temperature, pulse and respiration taken frequently during one of these attacks gave the following data:

TABLE 2.—Temperature, Pulse and Respiration Records (Case 2)

Time	Pulse	Respiration	Temperature
8 p. m.	148	34	99.8
9 p. m.	158	36	102.0
10 p. m.	152	36	102.4
12 p. m.	160	32	100.4
2 a. m.	148	30	99.6
4 a. m.	126	28	98.0
6 a. m.	132	28	99.0

During the month of August restlessness and whining, which characterized the latter part of her illness, became progressively more marked. While the patient was regaining partial use of the paralyzed limbs, a coarse irregular tremor appeared. During this time the elbow and knee jerks were exaggerated and the Babinski reflex and ankle clonus were elicited on both sides. A marked right clubfoot next developed, necessitating the application of a plaster cast which was kept on for two weeks. The left foot was only slightly affected.

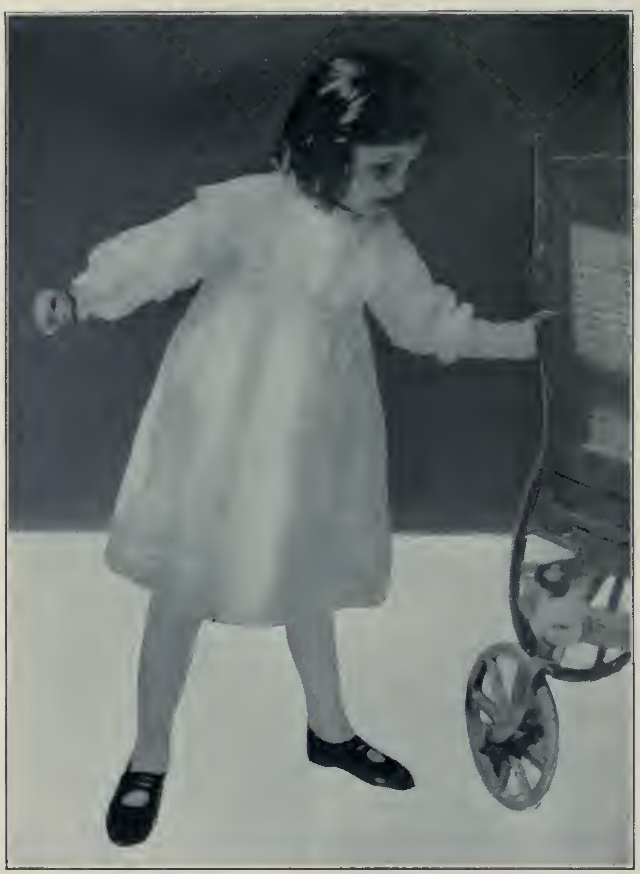

Fig. 2 (Case 2).—Appearance of patient the day she left the hospital.

By the latter part of August great improvement had begun. The pupils became regular, ptosis disappeared and the characteristic cry was changed occasionally to almost a speaking tone. At this time dysphagia disappeared to the extent that esophageal feedings were discontinued. During this period the patient exhibited rather striking modesty about being exposed. Even though her mental state was seemingly dull and she could not answer simple questions, she would always cover very promptly any exposed parts with her clothing or the sheets.

By the third week of September she began to take notice of what was going on about her. She nodded her head in answer to questions and took interest in her doll. At night she was frequently noisy and restless. By this time the facial palsy as well as ankle clonus and the Babinski reflex had entirely disappeared. Toward the end of September the patient's mind had brightened greatly, her special senses had returned to normal, and she was able to walk a little with assistance. She became more irritable and erratic, talking to herself and laughing, but creating disturbance on the slightest provocation.

Laboratory Findings.—Urine: Only July 4, the urine was cloudy, yellow, with specific gravity, 1.020; moderately cloudy from albumin; there were no sugar, a moderate number of granular casts, no indican and no acetone. July 13, and at the next three examinations, practically the same findings resulted with the exception of a faint trace of acetone. On August 15, there was a faint trace of albumin and an occasional hyaline cast. Before the patient was discharged the urine was entirely normal. The findings are summarized in Table 3.

TABLE 3.—BLOOD FINDINGS (CASE 2)

Date	Hemoglobin	Red Blood Cells	White Blood Cells	Polymorphonuclears	Lymphocytes	Mononuclears	Transitionals	Eosinophils	Basophils
July 5	48	2,850,000	5,600	47	46	3	2	1	1
July 24	56	2,920,000	6,100	58	34	4	2	2	0
Aug. 2	64	3,200,000	6,800	49	45	4	1	1	0
Aug. 21	..	....	8,300	70	23	5	1	0	1
Sept. 15	..	....	7,200	68	26	4	1	1	0
Oct. 1	78	4,050,000	6,900	64	29	3	2	1	1

Wassermann Test: The Wassermann reaction of the blood was negative. Widal Test: This test was done on July 18, August, 12 and September 28, with negative results each time.

Spinal Fluid: Spinal fluid examination yielded the results noted in Table 4.

TABLE 4.—SPINAL FLUID FINDINGS (CASE 2)

Spinal Fluid	July 5	July 24
Amount	20 c.c	11 c.c.
Pressure	+	0
Cell count	8	10
Nonne reaction	0	0
Noguchi reaction	++	+
Albumin	+	+
Fehling's	++	++
Bacteria	0	0
Tubercle bacillus	0	0
Wassermann test	0	0

The stools were negative. Plating on Conradi-Drigalski and Endo mediums showed no growth of typhoid bacilli. Throat and nose cultures were also negative.

The patient was discharged from the hospital October 3, and when last heard of, was almost entirely well, except for tremor of both upper extremities.

CASE 3.—*History.*—P. Q., male, aged 50, first complained of diplopia and of some difficulty in swallowing, February 21. At about the same time his family noticed that he was unusually quiet. On February 27, he gave up his work because of increased drowsiness, having fallen asleep several times at his bench in the factory.

Examination.—He was admitted to the Waterbury Hospital on March 2, the temperature was 100.8 F.; pulse, 84; respiration, 20. He was drowsy, and his face mask-like, absolutely devoid of expression.

The pupils were equal, regular and reacted to light and accommodation. There was left ptosis and moderate facial palsy. Speech was very slow, somewhat scanning, articulation and phonation normal. He answered questions very slowly, as if meditating, but quite accurately. On being ordered to raise his arm or leg he did so very slowly. There was a latent period between the command and the execution which could not be shortened, no matter how vigorously the order was given.

The tongue was coated, had no tremor and protruded in the median line. Pharynx, heart and lungs were negative. The blood pressure was systolic 128, and diastolic, 84. There was no rigidity of the neck, no tache cérébrale, no Kernig's sign. The knee jerk on the right seemed to be slightly increased and there was false ankle clonus on the same side. The cremaster and abdominal reflexes were normal.

On the day of admission, the white blood cells numbered 7,400; polymorphonuclears, 70; lymphocytes, 25; mononuclears and transitionals, 3, and eosinophils, 2 per cent. Urine: Specific gravity, 1.024; acid, yellow; no albumen or sugar. There was a slight increase of indican and a trace of acetone.

On the third day after admission 12 c.c. of cerebrospinal fluid were withdrawn; there was no increase of pressure; eighteen cells per c.mm. were found; polymorphonuclears, 16; lymphocytes, 76, and mononuclears, 8 per cent. Only fifty cells were counted, as there were very few cells in the smear. The Wassermann test was negative. Bacteriologic smears and cultures yielded negative results.

Creatinin and sugar determinations of the blood were made: creatinin, 1.4 mg. and sugar, 63 mg. per 100 c.c. of blood. Alveolar carbon dioxid determination by the Marriott method was 40. Blood Wassermann and Widal tests were negative.

Course and Treatment.—The patient's condition remained about the same for sixteen days. The temperature ranged between 98 and 101 F., the pulse rate between 74 and 126; respirations were never above 24. His most distressing symptom was difficulty in swallowing; he had to be fed very slowly and carefully in order to avoid distressing spells of coughing and choking. The ptosis was most marked on the third day, the patient being entirely unable to lift the eyelid. Internal strabismus was also present, but varied from time to time. There was no particular rectal or vesical disturbance in this case.

March 18, the patient's mental state was much brighter and he was able to swallow much better. The temperature, pulse and respiration were normal. The patient continued to improve rapidly and was able to sit out of bed during his fourth week in the hospital. The facial palsy disappeared quickly, but some ptosis persisted. The drowsiness disappeared entirely and he was discharged cured on April 6.

CASE 4.—*History.*—E. C., a girl, aged 5½, with a negative family and personal history, except for a mild attack of influenza in November, 1918, had malaise, occasional headache and general indisposition during the week of Oct. 20, 1919. Occasionally the child exhibited twitchings of the head and right side of the face. She was put to bed on October 25, and during the next six days had irregular fever, obstinate constipation, involuntary emission of urine and marked disturbance of mentality. At first she was markedly delirious and apparently had visual hallucinations, but soon the delirium was less constant and alternated with periods of drowsiness and lethargy. She became hard of hearing. There was also considerable dysphagia, and for two days prior to admission she took little nourishment.

Examination.—She was admitted Nov. 3, 1919, at 2 p. m., with a temperature of 102.2 F.; pulse, 100; respiration, 28. She was well nourished, quiet and apparently sleeping when left alone, but restless and tossing when an attempt was made to examine her. From time to time she uttered a peculiar cry, and when her eyes were opened, would stare about aimlessly. The pupils were equal, regular, dilated, reacted poorly to light. Mouth and pharynx, heart and lungs were normal. There was neither rigidity of the neck nor Kernig's sign; knee jerks and ankle jerks were normal; there was no Babinski reflex. Results of otoscopic and ophthalmoscopic examinations were negative. Throat smears and cultures were negative for diphtheria and for Vincent's angina.

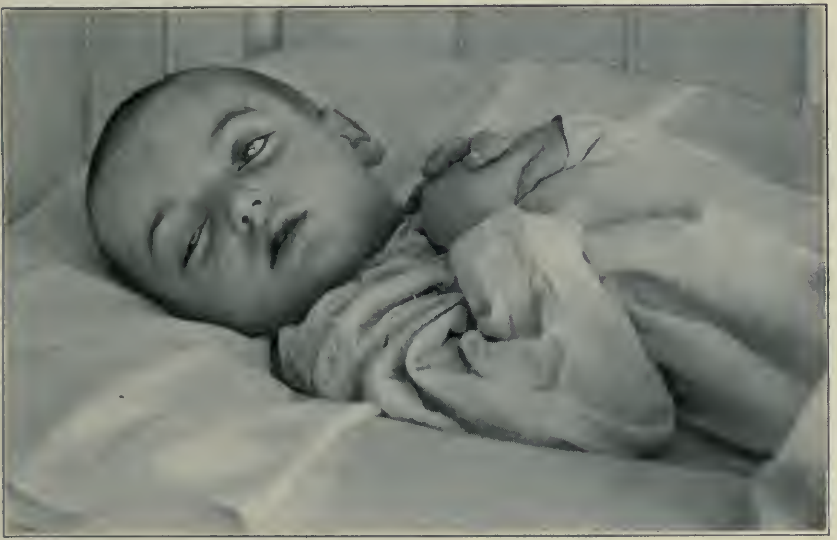

Fig. 3 (Case 4).—Appearance of patient, the fourth day after admission to hospital.

The white blood cell count soon after admission to the hospital was 15,400; polymorphonuclear cells, 74; lymphocytes, 18; mononuclears and transitionals, 7; eosinophils, 1 per cent.

Course and Treatment.—Three hours after admission the spinal fluid was under normal pressure, and clear, except for a slight tinge of blood. Globulin was slightly increased. The differential cell count was: polymorphonuclears, 4; lymphocytes, 78; mononuclears, 18 per cent. Smears and cultures showed no bacteria. A search for tubercle bacillus with the antiformin method was negative.

November 4: The delirium and restlessness were less marked; involuntary emission of urine and feces was reported; all nourishment was refused and feeding by tube three times daily was ordered. Physiologic sodium chlorid solution, 5 ounces per rectum, was given every six hours.

November 5: The von Pirquet and Widall tests were negative.

November 6: Wassermann on the spinal fluid was negative. Blood findings were: hemoglobin, 90 per cent.; red blood cells, 4,440,000; white blood cells, 16,200; polymorphonuclears, 75; lymphocytes, 17; mononuclears and transitionals, 7; basophils, 1 per cent. The carbon dioxid alveolar air determination gave 45.

November 8: There were ptosis on the right and facial paresis on the same side, also coarse tremor of arms and hands. The child changed her position but very little, being mostly supine with hands folded across her chest and fingers interlaced (Fig. 3). During the last forty-eight hours she was drowsy and lethargic most of the time. Deglutition was difficult, but she was able to take sufficient nourishment.

November 9: Peculiar vasomotor phenomena were observed during the last twenty-four hours. One half of the ear, for example, was flushed, while the other half was completely blanched. Flushing of one or the other cheek was frequent. The right hand was cold and blue, although the radial pulse was the same on both sides.

November 10: There was moderate rigidity of the neck and slight retraction of the head. There was a suggestion of Kernig's sign with exaggerated knee jerks, with ankle clonus and Babinski reflex on the left. Fiften c.c. of clear spinal fluid was withdrawn under slightly increased pressure. Cell count was 18 per c.mm. Differential: polymorphonuclears, 6; lymphocytes, 78; mononuclears, 16 per cent. Smears and cultures were negative.

During the rest of that day the condition remained about the same. The stupor was deep. She swallowed with great difficulty and at times perspired very profusely. Surface temperature was taken on the paralyzed and normal side of the face and over normal areas and those with vasomotor disturbances, but no definite differences could be detected. Toward evening the patient's condition became much worse. The respirations were irregular and Cheyne-Stokes in character. The pulse became progressively more rapid, but remained regular to the end. A pulse tracing two hours prior to patient's death showed no irregularities.

The patient died at 1 a. m., November 11, apparently with pulmonary edema as the terminal event.

Necropsy Findings.—Necropsy was performed eight hours after death by Dr. George M. Smith. There were no suggestive anatomic findings, outside of the brain. A specimen of urine obtained postmortem showed neither albumin nor casts, neither acetone nor diacetic acid, but 2.5 per cent. of sugar. Cultures taken from the intestines for typhoid bacilli were negative.

Examination of the brain revealed congestion of the meninges with considerable dilatation of the veins. Over the parietal region there appeared, here and there in the pia arachnoid, a small exudate, following the course of sulci. At the base there was intense congestion with a grayish yellow exudate of moderate degree in the region of the optic chiasm. Cultures taken from the exudate and from the cortex of the brain showed no growth. Smears showed red blood cells, lymphocytes, mononuclear cells and an occasional polynuclear cell. No organisms were found with methylene blue and acid fast stains.

On section of the formadehyde hardened brain, the ventricles appeared slightly dilated, but the ependyma and choroid plexuses were normal. Sections were made through the meninges and cortex of the brain and from various parts of the base. Sections from the cortex (parietal and occipital lobes) showed in each instance considerable involvement of the meninges. The blood vessels were dilated and there was an accumulation of inflammatory cells in the pia arachnoid consisting chiefly of lymphocytes and mononuclear cells. The cortical gray and subcortical white matter showed dilated vessels, and here and

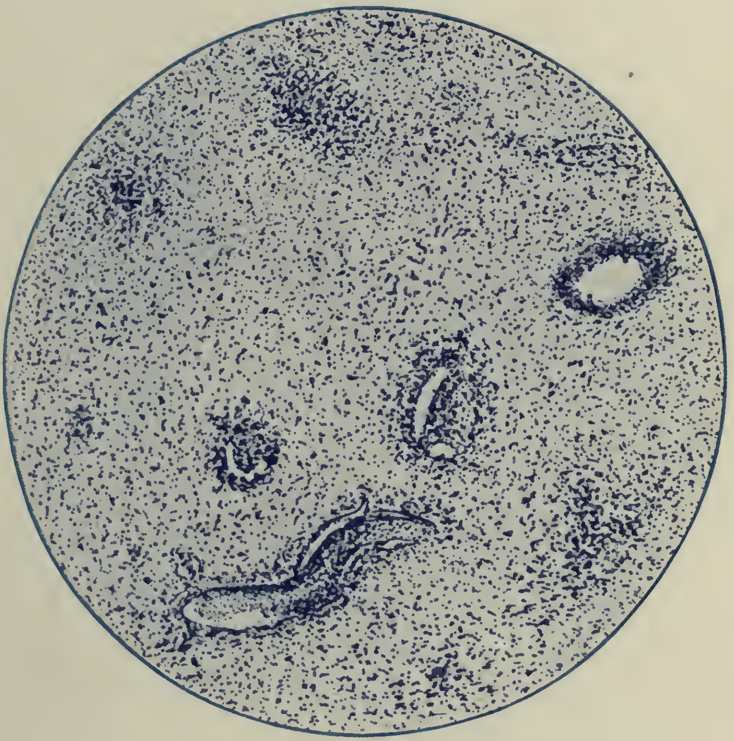

Fig. 5 (Case 4).—Midbrain, region of optic thalamus; capillary engorgement, dilatation of blood vessels and perivascular exudation.

there capillaries surrounded by a few lymphocytes. Sections of the base of the brain showed most marked involvement in the region of the optic thalamus. Here no hemorrhages were evident, but the capillaries were distended with blood. There was marked perivascular infiltration with lymphocytes and mononuclear cells. In places underneath the pia arachnoid and in the substance of the brain an accumulation of lymphoid cells was present. In other places there was a heaping up of endothelial cells of the pia directly in contact with the brain substance.

In the pons the vessels were congested, but the perivascular infiltration was less marked. In the medulla, at the floor of the fourth ventricle, the congestion was much more apparent. There were several hemorrhagic areas, the vessels and capillaries were dilated and surrounded by a perivascular infiltration of lymphocytes and mononuclear cells.

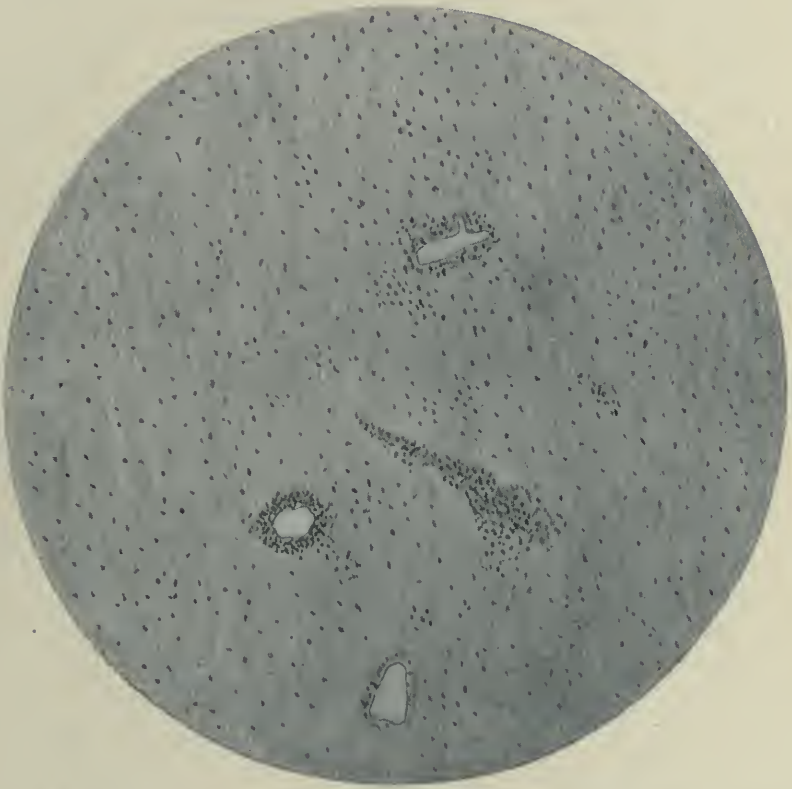

Fig. 4 (Case 4).—Region of floor of fourth ventricle; engorged blood vessels and perivascular infiltration. Diffuse exudate in gray matter.

ANALYSIS OF THE CASES REPORTED IN THE LITERATURE

In reviewing the literature on encephalitis lethargica the main difficulty lies in the uncertainty and inauthenticity of many of the cases, particularly those reported in the early part of the epidemic. It is evident that, even at this date, neither the etiology nor the clinical entity of the disease has been definitely ascertained. The Australian cases began abruptly with convulsions, and coma was a characteristic feature. In none of the cases was ophthalmoplegia present, and lethargy was not a feature except in those reported by Burnell.[1] Pathologically, the investigations of these cases were all fruitless except those reported by Breinl[2] who concluded that the pathologic findings justify the opinion that the cases were forms of poliomyelitis.

Since that time the consensus of opinion seems to have been that encephalitis lethargica differs decidedly from poliomyelitis and anything else that has been described. It is to be mentioned here, however, that very recently Loewe and Strauss [3] cultivated a virus obtained from cases of encephalitis lethargica on culture mediums, with the methods employed by Noguchi for the cultivation of spirochetes, and they report finding an organism very similar to that described by Flexner and Noguchi in poliomyelitis. The material for investigation obtained by these authors presumably is from cases reported by Sachs,[4] some of which are as typical as any that have been reported during the epidemic. Therefore the statement of the above investigators may be of great significance since it corresponds with the conclusions drawn by Breinl from his studies of the Australian cases. This would tend to indicate that the various forms of the "mysterious diseases" may be different forms of poliomyelitis.

For the present, however, it seems reasonable that the cases of peculiar lethargy and ophthalmoplegia reported during the last eighteen months represent a definite clinical entity hitherto unknown, unless we include cases that followed influenza epidemics in the past and have been known as Schlafkrankheit, Nona, Coma somnolentum and "catarrhal fever" with "soporosite."

We have attempted to analyze the clinical features and laboratory findings of all the cases reported in the current literature. A great many of these are vague, incomplete and atypical. For the purposes of this analysis we have taken the symptoms given by Bassoe [5] as typical of the disease, and have excluded all cases that totally differ from his description.

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1. Burnell, G. H.: M. J. Australia **2**:157 (Aug. 25) 1917.

2. Breinl, A.: Clinical, Pathological and Experimental Observations on the "Mysterious Disease," a Clinically Aberrant Form of Poliomyelitis, M. J. Australia **1**:209 (March) 1918.

3. Loewe, L., and Strauss, J.: Etiology of Epidemic (Lethargic) Encephalitis, J. A. M. A. **73**:1056 (Oct. 4) 1919.

4. Sachs, B.: Epidemic Encephalitis (Central or Basilar), N. Y. Med. J. **109**:894 (May 24) 1919.

5. Bassoe, P.: Epidemic Encephalitis (Nona), J. A. M. A. **72**:971 (April 5) 1919.

Excluding the cases reported by the special British government board[6] appointed to investigate this disease, we have reviewed 182 cases. From this number we selected 100 cases as being typical and more completely reported.

Age Incidence.—One case mentioned in the literature by Harris[7] may be accepted as congenital. [A woman suffering from undoubted encephalitis gave birth to a normal infant that seemed drowsy. On the third day lethargy developed. This lasted several days with complete recovery.] The youngest patient reported in the literature was 4 months; the oldest 62 years. Fifteen per cent. of the patients were below 10 years of age; 12 per cent. were between 10 and 20; 30 per cent. between 20 and 30; 19 per cent. between 30 and 40; 17 per cent. between 40 and 50, and 6 per cent. between 50 and 60. One patient was 62 years old. There were no age records in twelve of the 100 cases analyzed. The age incidence is shown in Table 5.

TABLE 5.—Age Incidence in Encephalitis Lethargica

Age in Years	Number	Per Cent.
Below 10	13	15
10-20	11	12
20-30	25	30
30-40	16	19
40-50	14	17
50-60	5	5
Above 60	2	2
Total	86	100

Sex Ratio.—Fifty of eighty-three cases, or 60 per cent., were males, while thirty-three, or 40 per cent., were females. The sex of the patient was not mentioned in seventeen cases.

History of Influenza.—This is reported in seventeen cases; in eleven the attack was one month prior to the encephalitis; in two, two months; in two, three months, and in one each, four and six months, respectively.

Symptoms.—Headache was mentioned as a more or less distressing symptom in 28 per cent. of the cases. In most of these it was an early sign of the illness. When lethargy was pronounced headache, of course, became subsidiary.

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[6] Report on an Inquiry into an Obscure Disease, Encephalitis Lethargica, Local Government Board Reports on Public Health and Medical Subjects, N. S. 121, London, H. M. Stationery Office.

[7] Harris, W.: Acute Infectious Ophthalmoplegia, Lancet **1**:568 (April 20) 1919.

Vertigo is mentioned in thirteen of the cases; like headache, it was frequently an early premonitory symptom and disappeared when the disease became well established.

Diplopia is mentioned in 33 per cent. and it was generally early; in many instances it was the earliest symptom. Its duration and severity varied greatly. In some it was so disturbing that the patient was obliged to keep one eye constantly closed. In most cases the diplopia persisted until the patient began to improve.

Ocular palsies were present in 57 per cent. The most common condition was ptosis of one or both lids. External and internal strabismus was next in frequency. Both ptosis and strabismus were frequently present. In a number of cases upward or downward motion was impaired, and some patients were apparently unable to move the eyeball in any direction.

Conjunctivitis is mentioned in five cases, and photophobia in the same number.

The pupils were equal and regular in the majority of instances. In a number of cases, however, inequality or irregularity of the pupil is mentioned. It seemed to bear no relation to the side of the face that was paralyzed. The pupils frequently reacted sluggishly to both light and accommodation, as particularly mentioned by Kennedy.[8] The Argyll Robertson pupil is mentioned in a case reported by Lortat-Jacob and Hallez,[9] also in one reported by Claude and Schaffer.[10] In both cases the Wassermann reaction was positive in blood and spinal fluid. The latter case proved fatal in spite of vigorous antisyphilitic treatment, and the histopathology indicated the diagnosis of encephalitis lethargica. Professor Netter,[11] in discussing the former case, said that in his opinion it was a case of encephalitis lethargica, and thought that an Argyll Robertson pupil may be present in this disease depending on the localization of the pathologic process.

In four cases choked disk is reported: in three engorgement of the retinal veins.

Dysphagia is mentioned in 17 per cent. of the cases.

Definite paralyses are mentioned in thirty-nine cases: in thirty-one, unilateral or bilateral facial palsy; in five, paralyses of the extremities; and in three, of the face and extremities. Mere weakening or partial paralysis of the extremities is not included in this classification.

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[8] Kennedy, F.: Epidemic Encephalitis with Stupor, Med. Rec. **95**:631 (April 19) 1919.

[9] Lortat-Jacob and Hallez, G. L.: Bull. No. 15-16, May 3, 1919, p. 639.

[10] Claude, H., and Schaeffer, H.: Encéphalite lethargique, Bull. et mém. Soc. méd. d. hôp. de Par. **43**:521 (May 29) 1919.

[11] Netter, A.: Bull. et mém. Soc. med. d. hôp. de Par. **43**:300 (April 4) 1919.

Tremors were present in 19 per cent. This was coarse and jerking in the majority of instances; in some it was choreiform. Ataxia, as evidenced by the finger-nose test, was present in many cases. Rigidity and catatonia are frequently reported.

There was an abnormal state of mind in 86 per cent. of the cases. In eighty-three it varied from slight drowsiness to profound stupor. It is variously described as listlessness, somnolence, drowsiness, lethargy, emotional stupor and mental inertia. While in some instances the lethargy was so profound that the patient could not be aroused, in the majority of cases the mind behaved lucidly under stimulation. The patients answered questions accurately, but only after some delay and with evident effort. They volunteered no information and fell back into apparent sleep after questioning was stopped. In three cases there was active delirium without lethargy. In eleven cases both lethargy and delirium were present during some period of the illness.

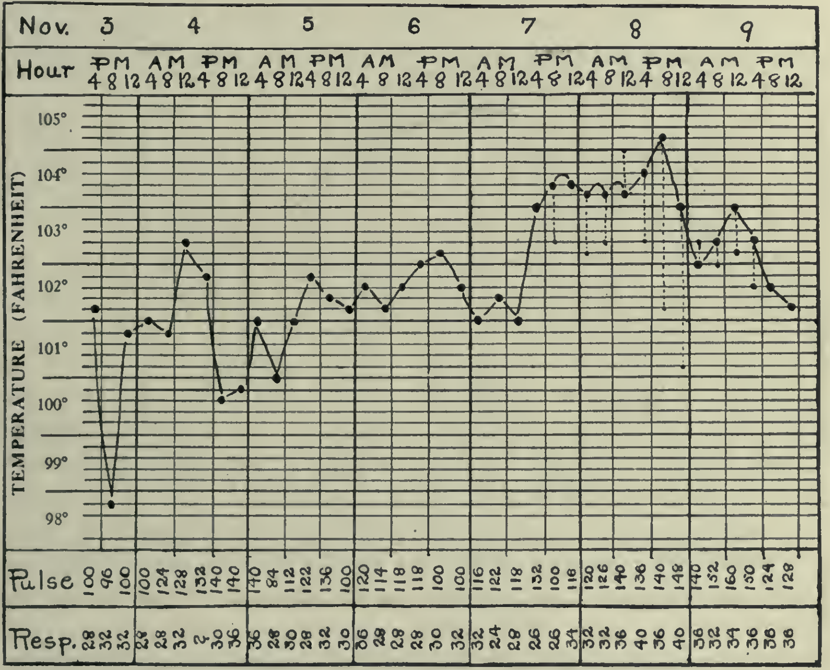

Fig. 6 (Case 4).—Temperature chart.

The face generally gave the impression of listlessness and was devoid of emotional expression. It is most frequently compared to that of paralysis agitans, with mask-like, blank, immobile features. Kennedy speaks of one case with the facial expression of advanced myasthenia gravis.

The reflexes were normal in twenty-eight cases; exaggerated in twenty-one and markedly diminished or absent in nine; a positive Babinski and ankle clonus are reported in seven cases; in twenty-four, no mention of reflexes is made. There was incontinence of urine and feces in 17 per cent. of the cases, while retention of urine and obstinate constipation are mentioned frequently.

The temperature was normal in six cases; in twenty it was less than 100 F.; in nineteen, between 100 and 102; in thirteen, above 102. In thirty-seven the temperature is not recorded.

The pulse was reported as normal throughout the illness in seven cases; below 100 in nine; between 100 and 120 in six; between 120 and 140 in nine, and not mentioned in sixty-seven cases. The respirations were generally but slightly affected.

The urine was normal in twenty-seven cases. In one case there was a faint cloud of albumin, in another, a few granular casts, and in a third albumin and casts were found; all of these cases ended fatally. We do not think it is of any special prognostic value, however, because a great many of the cases that ended fatally did not present any abnormal urinary findings. Case 2 of our own series showed albumin and casts on several occasions, but ended in recovery. The absence of acetone was mentioned in two cases, and its presence in one case.

There are very few hemoglobin and red blood cell reports in the literature. Where such findings are reported, the results are normal or show evidence of slight secondary anemia. Forty-three white blood counts and eighteen differential counts are recorded. Out of the forty-three reported, two counts are below 6,000; twenty-eight between 6,000 and 10,000; nine between 10,000 and 15,000; two between 15,000 and 20,000, and two are above 20,000. In 70 per cent. of these cases the white count was below 10,000, and it was below 15,000 in 90 per cent. The polymorphonuclear count was generally normal or slightly diminished; it was above 80 per cent. in only three cases. The mononuclear cells seem to have been quite conspicuously increased in number. They were above 4 per cent. in thirteen of the eighteen differential counts recorded, and above 8 per cent. in eight of the cases. A slight increase of the eosinophils is reported by several authors.

The Wassermann reaction is reported positive in two cases and as ± in three cases. As mentioned in connection with the consideration of pupillary reactions, the cases are here included because the clinical histories indicate that they were true cases of encephalitis with possibly coincidental syphilitic infection.

The blood cultures were negative, with the exception of one case in which the pyocyaneus was obtained.

The spinal fluid findings are reported in seventy-nine of the 100 cases analyzed. There was a slight or moderate increase in sixteen; the fluid was mentioned as cloudy in two cases. The cell count was less than 20 in forty-two cases, between 20 and 50 in eleven, between 50 and 100 in six, and above 100 in three cases. Globulin was slightly increased in seventeen, and is reported as normal in sixty-two cases. The differential count is given by Pothier [12] as all lymphocytes and mononuclear cells, with the exception of one case in which two polymorphonuclear cells are reported. Similar findings are reported by Claisse [13] and other French authors, and also by Wedgeworth and by Bassoe.[14] Wilson [15] reports one case with 90 per cent. polymorphonuclear cells, but in this case there was evidently some complication, as is evidenced by a blood count of 33,000 leukocytes with a differential count of 92 per cent. polymorphonuclears.

The Wassermann in the cerebrospinal fluid is reported as negative in all the cases where the test is mentioned, with the exception of the two cases referred to above.

The bacteriologic findings were uniformly negative with the exception of one case reported by Stafford.[16]

Termination. — Twenty-three of the cases analyzed terminated fatally; in forty-five recovery was complete, and thirteen are reported as recovering. Nineteen of the 100 cases were reported during the course of the illness and the termination is therefore not recorded. Some authors report very low mortality rates, as for example Hall,[17] who had no deaths among his sixteen cases. Abrahamson [18] reports a mortality of 10 per cent., while Wegeforth and Ayers,[19] and others, report a mortality rate of 40 per cent. or higher.

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[12] Pothier, O. L.: Lethargic Encephalitis, J. A. M. A. **72**:715 (March 8) 1919.

[13] Claisse, P.: Encéphalite lethargique, Bull. et mém. Soc. méd. d. hôp. de Par. **43**:222 (March 7) 1919.

[14] Bassoe, P.: Epidemic Encephalitis (Nona), J. A. M. A. **72**:971 (April 5) 1919.

[15] Wilson, S. A. K.: Lancet **2**:7 (July 6) 1918.

[16] Stafford, C. M.: Encephalitis Lethargica, J. Lab. & Clin. Med. **4**:691 (August) 1919.

[17] Hall, A. J.: Brit. M. J. **2**:461 (Oct. 26) 1918.

[18] Abrahamson, I.: Epidemic Polioencephalitis, New York M. J. **110**:17 (July 5) 1919.

[19] Wegeforth, P., and Ayer, J. B.: Encephalitis lethargica, J. A. M. A. **73**: 5 (July 5) 1919.

FURTHER OBSERVATIONS ON NEUROSYPHILIS AND THE PSYCHOSES *

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Syphilis of the nervous system has been attracting considerable attention from syphilographers of late, so much so that at the present time the journals are full of reports dealing with spinal fluid findings in cases of secondary and latent syphilis, in many cases with remarkable serologic evidence of invasion of the central nervous system with or without definite neurologic signs. Many represent latent cases of paresis or cerebrospinal syphilis, in which the invasion by spirochete and alterations in the spinal fluid occur a considerable time before we have any clinical evidence of such invasion. Cases of this type are much more frequently seen in the outpatient department of general hospitals, or in the practice of a syphilographer, than they are in nerve clinics or in the outpatient department of the psychopathic hospital. As mental symptoms are usually absent, such cases are only rarely seen in the state institutions.

On *a priori* grounds we should, I think, expect to find a great many more mental cases than we actually do, with laboratory evidence of syphilis of the nervous system and a psychosis of a type not usually associated therewith. This expectation is based on the fact that so many cases are now being reported in which, despite the absence of any clinical symptoms at all, there is laboratory evidence of invasion of the central nervous system during the secondary or somewhat later stages of syphilis. In mental cases, however, it has been amply shown in statistical and other reports on syphilis, Wassermann reactions and spinal fluid examinations, that this does not frequently occur, and we find only rarely cases that, for example, show a dementia praecox picture and have the serologic findings of neurosyphilis. That this is not due to the fact that syphilis does not occur in dementia praecox, is shown by my own observations that 5 per cent. of 240 cases of dementia praecox showed a positive Wassermann reaction in the blood serum (spinal fluid examinations negative), and this finding is amply borne out by the statistics of other observers at other institutions. Of importance, also, is my further finding that among 1,500 nonparetic, noncerebrospinal syphilis cases admitted at Danvers State Hospital, some 7.5 per cent. gave a positive Wassermann reaction in the blood serum; but very few of these showed also any changes in the spinal fluid. Accordingly, we must adhere to the view that mental symptoms associated in a given case with signs of invasion of the central nervous system by syphilis must be attributed to the latter. If this were not true, then we should expect to find relatively large numbers of cases showing heterogeneous types of mental symptoms associated with the serologic findings of neurosyphilis, just as we now find so many cases without any symptoms whatever associated with the serology of neurosyphilis. The mechanism of the causation of these atypical cases must remain in doubt until a sufficiently large group has been collected and subjected to rigorous clinical and postmortem examinations to make the relationship clear. In some cases, granted that the association is that of cause and effect, it is fairly clear that the psychotic symptoms are the result of a particular disease agent acting on a particular type of personality.

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In a paper published in 1917, I reported eleven cases of neurosyphilis showing divergence in the clinical and serologic pictures. Since that time Southard and Solomon have published a book[2] in which they have presented a considerable group of such atypical cases. Smith and I have published a paper,[3] dealing with two cases of degenerative chorea in which the serologic findings of general paresis were present, one of these having been discovered since the publication of the first paper mentioned. Myerson[4] has also presented a group of atypical cases.

Hoch[5] recently reviewed my article, pointing out that certain of the cases therein were probably examples of that type of psychosis associated with neurosyphilis, first described by Plaut and called by Kraepelin "syphilitic paranoid condition." Shortly after I published my previous paper I became aware that such a group had been described. While it is difficult to be entirely certain in the cases quoted in my earlier paper of the relationship to this group of cases, I am

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1. Lowrey, L. G.: Some Observations on the Relationship Between Neurosyphilis and the Psychoses, Am. J. Insan. **74**:25 (July) 1917.

2. Southard and Solomon: Neurosyphilis, Boston, W. M. Leonard, 1917.

3. Lowrey, L. G., and Smith, C. E.: Two Cases of Chorea (Huntington's Type) with the Serology of General Paresis, Am. J. Syphilis **2**:453 (July) 1918.

4. Myerson, Abraham: Five Cases of Neurosyphilis Illustrating Special Points in Symptomatology and Course, Boston M. & S. J. **180**:499 (May) 1919.

5. Hoch, A.: Abstracts from Current Literature, Arch. Neurol. & Psychiat. **1**:642 (May) 1919.

inclined to agree with Hoch in his placing of certain cases there presented.

The cases to be presented at this time fall into certain definite groups. Group I, comprising six cases, represents a group of paranoid psychoses. Cases 1 and 2 possibly represent the above mentioned type of syphilitic paranoid, although in each case we have a history of the use of large amounts of alcohol preceding the onset of the psychosis. In Case 3 a paranoid personality seems to have developed into a mild psychotic state under the influence of neurosyphilis. Case 4 developed an interesting transformation of the personality in the midst of a typical paretic excitement, thus apparently confusing the issue with respect to the paranoid types of neurosyphilis.

Cases 5 and 6 are very obscure. In each case we deal with a paranoid condition of rather unusual type associated with serologic findings which are not conclusive evidence of neurosyphilis. The propriety of including such cases may be questioned, but since they illuminate points in connection with the discussion of the relationship between paranoid psychoses and neurosyphilis, and in connection with the diagnosis of neurosyphilis, they have been included.

In Group 2 there are five cases of the manic depressive reaction type, in Case 11 the diagnosis of neurosyphilis being uncertain. Case 10 is clearly not an association of manic depressive psychosis with neurosyphilis, but a paretic case in which intensive treatment produces remissions of an extraordinary type—justifying inclusion with the group. Group 3 contains two feebleminded cases; one with serologic findings of neurosyphilis, probably congenital; the other with serologic findings of probable neurosyphilis. Group 4 contains four cases with neither psychotic symptoms nor clinical evidence of neurosyphilis. In three cases there was conduct disorder, probably determined by so-called constitutional inferiority. In Case 16, there was an interesting increase in serologic findings under treatment.

In addition to these groups there is one case showing increase in serology during treatment, and one case, by way of contrast, of pseudoparesis.

Analysis of the clinical signs and symptoms of neurosyphilis shown in all nineteen cases, reveals the interesting fact that in only four cases could the diagnosis of neurosyphilis be made without the evidence of the lumbar puncture and Wassermann reactions. In one additional case an improper diagnosis of neurosyphilis could be refuted only by such evidence. In four cases, the clinical evidence is nil and the serologic evidence equivocal. Accordingly, of the fourteen cases of undoubted neurosyphilis here reported, only four, or 28 per cent., present clinical evidence of the syphilitic condition present. To be sure, the cases in the group are unusual from any point of view, but the inevitable conclusion to be drawn is that any case with nervous or mental symptoms of any type may have nonsymptomatic or atypical symptomatic neurosyphilis, and all such cases should, in the absence of contraindications, receive lumbar puncture.

CASE HISTORIES

Group 1. Paranoid Cases

Case 1 (File number 10847).—*Alcoholic, drug using prostitute, a victim of conjugal neurosyphilis. Psychosis characterized by auditory hallucinations, delusions, depression, hypokinesis; clearing to an hallucinated state, with defective judgment and considerable reaction to hallucinosis. Treated.*

History.—A woman, aged 34, was sent to the hospital in 1918 with the statement that for two weeks she had had delusions that men were coming up the front steps and talking about her, saying they would enter her room and kill her. The patient said she had been ill since December, gradually becoming depressed and of late had heard voices. She admitted the use of morphin, heroin and cocain for seven years, and the use of considerable quantities of alcohol for at least fifteen years. Her responses to questions were very slow; she moved very little and then very slowly. She would answer very few questions regarding her hallucinations. At times her lips would tremble, as though she were trying to speak, but words would not come. She was worried and felt depressed. She recognized the voices, but could not tell whose they were or what they said.

A history was obtained from her husband, who was recognized to be a paretic from his memory defect, speech defect and expressionless face. (Later the husband died in the hospital of paresis, having been under treatment for some months.) He said that the patient had been a prostitute since the age of 16, continuing so during her married life. They had been married for ten years, for two years of which the husband did not work. She had used alcohol for many years, going on sprees once or twice a month. He agreed that for about seven years she had used drugs. He noted the first change in the patient about Easter time, 1918, when she remained in bed one day and said she was gone, that she would not live any more, and might as well take poison. She then began saying that people had no use for her and were going to kill her. The voices would say, "She is no good, she is rotten." She had some swelling of the leg, but from this she recovered. She was blue, depressed; would sit or stand for hours and was very inactive. The husband threw away the drugs and she had had none for some time before entrance.

Examination.—Physical examination revealed nothing remarkable. The tongue and hands were slightly tremulous. The facial lines were obliterated. All reflexes were normal, including the pupils. There was marked cyanosis of the hands, with blanched nails. There was a considerable degree of pyorrhea.

Course and Treatment.—She had hallucinations, hearing voices threatening her, calling her bad names, and accusing her of all sorts of immoral practices. When she became slightly more accessible she gave a history of gonorrhea and syphilis, and admitted having had hallucinations in 1917. Her memory was only fairly good. The speech was hesitant and much retarded.

Questions frequently had to be repeated. She believed that people spied on her; published vulgar things in the paper; that they had tried to poison her. She was certain that she must die. A voice accused her of all kinds of sex perversions, which she admitted were true. She claimed to be depressed, but seemed really to be rather indifferent to her painful ideas. She did not answer the voices directly. There seemed to be blocking, especially regarding the content of her hallucinations. This condition continued for about three months, during which time she was under intensive antisyphilitic treatment. She then gradually became lively and alert, noticing everything that went on around her. She had hallucinations constantly, the voices often saying terrible things to her, many times so absurd as really to be amusing She referred her hallucinations to people who were going along the road outside the hospital. She was somewhat silly, and at times was quite elated. Occasionally she would become very profane in reaction to the voices. Very frequently she would suddenly remark, "It is not true, doctor, what they say about me; I didn't do that," indicating that the voices did not talk to her directly, but talked about her. She had some insight into her past condition, but thought she was well at the time. In the later months, there were no longer any tremors or any cyanosis.

She was eventually transferred to the Boston State Hospital where she now is. Active hallucinosis has continued, despite treatment.

At the time of admission the Wassermann tests on blood and spinal fluid were positive. In the spinal fluid the protein was increased; there were sixteen cells, and a gold reading 5554444322. There were tremors of the hands and facial muscles, and an ironed-out expression. These latter symptoms, plus the serologic findings, were the only positive symptoms of syphilis of the nervous system. The other symptoms were more typically those of an alcoholic hallucinosis, or of dementia praecox, and in the absence of serologic findings the differential diagnosis between these two would be very difficult. A recent spinal fluid examination showed a lessening in the intensity of the reaction.

CASE 2 (File number 13430).—*An alcoholic man, aged 36; acquired syphilis eighteen months ago; vigorous treatment. Psychosis characterized by vivid, threatening auditory hallucinosis with delusions of persecution. Wassermann reaction on blood and spinal fluid, negative. Serology otherwise typical. Treatment—recovery from the psychotic state in a few weeks.*

History.—The patient, a man of Irish descent, was brought to the hospital by the police, with the statement that he had been bothered by the influence of neighbors, who had some sort of an instrument by which they could read his thoughts, control him so he could not talk, etc. This instrument enabled them to see right through the walls. He stated that this had been going on for six months. He seemed quite concerned about his persecutions. His emotional tone was in accord with the delusional content. He was accessible and cooperative, with no memory loss. He said, "I can't dope it out. I know it sounds like a crazy man, but these people can make a suggestion and then make me carry it out. When I was in Maine they had me in a hypnotic trance."

History obtained from the wife revealed nothing of importance in the family history. The patient had had some education, and later became a bartender, at which business he had worked until about five years ago, when he became an advertising solicitor. He had been married for thirteen years. His wife, during the past seven or eight years, had worked in order to help support the family. She stated that he had always been jealous, although she gave no cause. He had a cheerful, easy-going disposition, was good-natured, generous, thoughtful, proud, did not like criticism. He read a great deal. Although he was ambitious, he never realized his ambitions. He had always drank a little, but more during the past winter, when he would become intoxicated. He stopped drinking May 1. He turned against his wife last spring and has had no marital relations since. He became irritable.

In July, he first stated that people were after him, and since that time he had complained of delusions of persecution. He finally refused to speak to any of the neighbors, thinking they were hired to annoy him. At times he thought that his wife was behind the plot. Once he threatened to kill his wife, his son and himself, but made no attempt. For about two weeks he smiled to himself, and his lips moved as though he were talking. He would start to answer questions and then hesitate. He complained of pain in his head.

On the day of admission he was yelling, saying that the roof of his head was going.

The patient himself gave a history of excessive alcoholism extending over a period of several years, and a definite history of a primary syphilitic lesion a year and a half before. He received active antisyphilitic treatment at a general hospital. The Wassermann reaction became negative and remained so.

On mental examination, he was oriented and there was no history of disorder of consciousness. There was no obvious disintegration of the personality; no memory loss. The emotional tone seemed quite in keeping with the delusional content. He was worried because of the persecutions. He claimed to feel depressed, and had outbursts of irritability against people who he thought might be in league with his persecutors. His hallucinations and delusions commenced six or seven months before entrance, following a spiritualistic seance which he attended in May. His hallucinations were auditory; the phonemeta were about him, referring to him in the third person, but occasionally the voices talked to him directly. They were chiefly catholics. What the ultimate motive was, he could not determine. They had some sort of an electro-magnetic machine which effected him through sound waves. The persecutors could control his thoughts and every activity. His will was under their complete domination. He was quite accessible and cooperative, and showed no conduct disorder. He was normally active, and there was no disorder of thinking.

Examination.—On physical examination, he was found to be well developed and well nourished. The pupils were equal, regular in outline, and reacted normally to light and accommodation. All deep reflexes were present and brisk. There were no abnormal reflexes. General visceral systems normal. The Wassermann reactions on the blood and spinal fluid were negative to several tests. Spinal fluid showed protein increase, 8 cells, and a gold reading 5555321000.

Course and Treatment.—Within six weeks of his admission, the patient had entirely recovered from his psychosis. He stated that his past experience had been a vivid dream, and claimed that the voices he heard and the ideas of persecution were all due to his imagination. He was pleasant and agreeable, quite tractable. He worked well and assisted with the general work around the hospital.

He had, in the meantime, received very vigorous intravenous and intraspinal treatment and at the end of six weeks was allowed to go home on a visit, where he still is, getting along quite well.

Without the complication of neurosyphilis, this would be regarded as a case of acute alcoholic hallucinosis. The patient's history of having used alcohol excessively up to a quart of hard liquors a day, combined with the onset of the psychosis shortly following the time when he stopped drinking, as well as the clinical characteristics of the psychosis, would all indicate this diagnosis. The ideas of influence, however, and of control were unusually marked for an alcoholic hallucinosis case. It should be noted that the syphilitic paranoid conditions are very closely allied in symptomatology to alcoholic hallucinosis, and that in both disorders we have to do with an active hallucinosis on which are founded delusions. Of course, in this case the diagnosis of neurosyphilis would be equivocal were it not for the clean-cut history of a primary lesion from a general hospital with resulting treatment, which produced a negative Wassermann reaction.

This case also emphasizes a point for the syphilographers, namely, that a negative Wassermann reaction in the blood serum is not always evidence of a thorough cleaning up of the syphilitic infection. As will be shown in another case, the negative Wassermann reaction in the blood serum may persist for a long period during which the spinal fluid findings are positive and may even become more marked. It may be noted here that the writer has had personal experience with one case in which the blood and spinal fluid Wassermann tests were persistently negative over a period of three years, although all the other spinal fluid findings were positive and the case was typically paretic from a clinical point of view. Eventually, under intraspinal treatment, the spinal fluid Wassermann test became faintly positive.

CASE 3 (File number 10373).—*A man, aged 42, of paranoid personality, with syphilis possibly four years previously, became quite suspicious and mildly psychotic. Improvement under treatment.*

History.—This man was referred to us from a general hospital because of certain paranoid ideas, together with a positive history of syphilis, leading them to suspect syphilis of the nervous system. In 1898 the patient's right eye was injured by the explosion of a fire-cracker. In 1906 he had been operated for double hernia and appendicitis. In February, 1916, he fell from a platform and injured his back. Roentgenograms taken at a general hospital led to the patient's being told that he had a slight fracture of the fifth lumbar vertebra. In 1915 he had gone to a general hospital for a sore throat and a sore on his right shin. A positive Wassermann reaction was found, and he received five doses of arsphenamin. At the time of entrance, March 14, 1918, the patient complained that his back and left arm felt numb, and that he was troubled with headache.

The mental examination showed that the patient was clearly accessible. He gave a good history of his family and of his own life. He had worked in an express office for many years, remaining twenty years in one office. He remained there so long because he lived with his mother and did not care to leave her. From 1911 to 1913 the catholics in this town were against him because he was a mason, and tried to run him out of town. He thinks they eventually succeeded in ruining his business, so he came to Massachusetts in 1913, and took a place in Boston in an express office. Here the men in the office soon began to make him the butt of their jokes and they began to do many things which irritated the patient: they would throw things at him and would take his chair from under him. More annoying even than this was the way in which they would change chairs for him. When he was out his chair would be taken and another one substituted. Many things of this sort were done, and he believes they talked about him and reported him to the foreman. He was quite circumstantial in telling this story. No disorder of articulation or of emotions appeared. He was quiet and cooperative. He described himself as having always been social, not seclusive or shy, easily angered, always the butt of jokes.

Examination.—His headaches, backaches, numbness and pains in his legs continued, and the physical examination showed an injured eye, a small mass in the occipital muscles, a stiff lumbar spine without curvature, without any pathology in the neurologic condition. The urine was negative. The spinal fluid showed globulin 1, albumin 2, gold 0012333100. The Wassermann reaction on the serum was positive, on the spinal fluid negative.

The patient has since been under treatment with improvement in the serologic findings. He has frequently visited me to talk about his complaints of one sort or other, usually physical, and has manifested a great deal of interest in the state of his health. He always asks for diet lists, and as to the advisability of his doing various things which he feels might in some way impair his health. To some extent the petty persecutions in the office have stopped. Whether this represents a lessening of the very mild paranoid strain, I cannot say. He is apparently not quite so suspicious of people as formerly, for when he was in the hospital he was definitely suspicious of many people whom he saw.

This case possibly represents a very mild type of the syphilitic paranoid condition described by Kraepelin, in which, however, hallucinations are absent.

Case 4 (File number 11177).—*Typical case of general paresis, developing marked auditory hallucinosis and transformation of the personality.*

This case differs from the others in that this man presented all of the clinical symptoms of general paresis, including Argyll Robertson pupils, hyperactive deep reflexes, slight speech defect, defect in calculation ability, judgment defect in practical matters, ideas of grandeur, restlessness and memory defect, together with the following serologic findings: Wassermann reaction, blood and spinal fluid, positive; protein increase, 12 cells and a gold reading 5555444321

Why then should such a typical case of general paresis be presented in a series such as this? For the following reasons: At the time of his first admission in August there were no hallucinations and had been none. He was out for four days on a visit in October, during which time his behavior was typically that of a grandiose paretic, and shortly after his return he became more and more restless, irritable and even violent, and about this period became actively hallucinated. He heard God's voice telling him that the physicians and nurses were to be killed. He also heard the voice of Jesus Christ and carried on long conversations with God and Christ somewhat later. The hallucinations continued, and he became convinced that he was Jesus Christ and damned everyone in his vicinity to hell, and later still came the idea, of variable intensity, that he was God. From the time of his admission on he had been under intensive treatment, and at one time developed a jaundice which interfered with the treatment for some weeks, during which period he became hallucinated. During all of this excitement and great expansion of the personality he still recognized those about him.

I cite this case because of its importance as showing that hallucinations can and do exist in general paresis, and that delusions, based thereon, may also occur; in addition to its bearing on the general question of the causal relationship between neurosyphilis and paranoid states, where the two co-exist. *If such a state as this may occur in otherwise typical paresis, then obviously neurosyphilis should be able to produce such a state without concomitant distinct signs of paresis.*

CASE 5 (File number 13364).—*A school teacher, aged 49, always queer, with a love fantasy dating back twenty years, and a paranoid psychosis of some years' duration, showing a curvature of the spine, possibly tuberculous in origin, with equivocal spinal fluid findings.*

History.—The patient was sent in by order of the court for observation. At the time of admission she was mildly excited by the experience, but showed an intelligent understanding of the situation. She stated she had had two operations within two months, and three days before had left a convalescent home. She denied any ideas of reference, delusions of all kinds and hallucinations. There was no dizziness, headaches or seizures. She was a teacher, lived alone, and for the last twenty years had worked irregularly.

Outside history was obtained with some difficulty, but it appeared that for many years she had been kept on the list of substitute teachers, because her work was not very satisfactory and she was hard to deal with. She was always dissatisfied with the way she was treated, and was antagonistic toward the children. In high school, the patient had been regarded as rather stupid, quiet and a little odd. She had taught school regularly for six years, and then as a substitute for twenty years. When she stopped her regular work, twenty years before, she ordered a trousseau from one of the stores and announced that she was going to marry a physician, who had never heard of her. Since that time she had been regarded as queer, but no one had thought that she had especially deteriorated. She had been known for rather queer religious beliefs, and for belief in spiritualism, and had told of visions and of illuminated pictures, but did not mention voices.

Twenty years ago she was at the Boston State Hospital for ten weeks, where a diagnosis of hebephrenia was made.

Examination.—On mental examination, the patient was courteous, very cooperative, but not freely accessible regarding her delusional ideas, which she tended to regard as sacred. From her, we learned that she was an illegitimate child, that she was cared for by her maternal grandparents until the age of 5, when an aunt by marriage took her and abused and mistreated her. She was then taken by some people who became her foster parents. She really knows very little of her family. As a child she was happy. She went through normal school. Her memory, in general, was good. School knowledge was well retained. Although she had taught for many years, she found it rather hard as the discipline was difficult for her. She regarded herself as a recluse. Diffident and bashful as a girl, easily embarrassed, she made very few friends. She did not mix well. She never cared for ordinary amusements; had never gone to shows or theaters; did not care for dancing, cards or sports of any kind. She called herself a bookworm. She usually read books on sociology and religion. She had never been subject to blue or depressed periods, and was not quick tempered. She was brought up a Baptist, but had changed her religious views since.

At about the age of 12 it was noticed that she had a curvature of the spine. At 14 a plaster cast was applied, and she wore it about three months. Later she had a support and a shoulder brace, which she had worn most of the time since. Eight years ago a small tumor was removed from the right breast. Six weeks ago another tumor was removed, and later, because of excessive menstruation, a curettage was done. She insisted strongly on returning home, and did so. The doctors visited her at her home and sent her here for observation.

The central feature of the case was a delusional idea which the patient gave up with much reluctance. She insisted that the examiner and every one in the universe knew about that with which she had to contend. If the examiner did not know, then she did not know and the whole thing would be imaginary. Twenty years ago something took hold of her and she knew she was loved. She had a wedding dress made, and mentioned having her name changed, and ever since then she had been influenced and had known that she was loved. This kept her from holding positions in the schools, and so influenced the pupils that their behavior was bad and she could not discipline them. This caused her all the misfortune and sorrow she had had, the climax being reached when she was sent to the psychopathic hospital. During the past two years this person had come to her every night and had had sexual intercourse. She enjoyed it, yet she worried about it because she was not sure that she was doing right. If she were in a repellant state of mind, he did not come.

Up to the age of 14 the patient performed masturbation, having been taught improper actions at the age of 5 by a boy. She overcame this and had not performed masturbation since. The patient would like to have married, and would like to have had a family. She thought that she was always hypersexual when around men. She felt that every one knew this. People looked at her on the street and noticed her. She could not give any reason as to why she thought this was known. She supposed there had been mention of it in the newspapers. She knew where the daughter of the man lived, but would not tell. Aside from this, no delusions were determined; nor was there any certain hallucinosis. She had a religion all her own. Her left hand was consecrated. The thumb signified the right spirit; the finger the right mind; the next the right body and environment; the next her relation with the Supreme Being and others; and the next the right consciousness. The right hand was named "service." The thumb indicated the spirit world. The fingers were called the family. She prayed according to the way she had named her hands. She was a socialist, and believed in spiritualism to a certain extent. She showed no disorder in thinking.

On physical examination she showed some dulness in the lungs; a right upper thoracic scoliosis. Neurologic examination was entirely negative. The blood and spinal fluid gave a negative Wassermann reaction. The spinal fluid showed increased protein, 3 cells, and a gold reading 5555511100.

Here, then, we have a patient always seclusive and odd with a definite sense of inferiority, due to the fact that she was an illegitimate child, with a diagnosis of hebephrenia twenty years ago at a State Hospital. She had scoliosis and possible tuberculosis of the lungs. She was sent to the psychopathic hospital because of delusions, chiefly of a sexual nature and of a spiritual lover. The Wassermann reactions were negative, but there was a typical paretic gold curve and an increase in the protein content. The cause for the spinal fluid findings is obscure, and the relationship between these findings and the psychosis is still more obscure. The chances are, of course, that there is no relationship, and that the organic disease indicated by the spinal fluid findings has yet to produce symptoms.

How are the spinal fluid findings to be explained? The positive findings were: albumin and globulin increase and the gold reaction. In the absence of clinical signs to aid us, we can only say that such findings indicate an inflammatory reaction in the nervous system. Such a gold curve is often regarded as typical for paresis, but this is not invariably true. While a negative Wassermann reaction in the blood and spinal fluid are not conclusive evidence against syphilis or paresis, it is a very rare finding. Furthermore, provocative arsphenamin injections produced no change in these reactions. Under the circumstances, we can only record the case and await developments.

Case 6 (File number 12636).—*A single woman, aged 51, apparently always reserved, and possibly somewhat peculiar, with some character change and economic failure four years previously. Psy-*

chosis very mild in type, characterized by probable auditory hallucinosis, and some delusional formation based on that. Patient anemic. Slight spinal fluid changes, probably not neurosyphilitic, but origin uncertain. Rapid improvement in mental condition. Discharge. No antisyphilitic treatment.

History.—The patient was admitted to the hospital with the statement that for six weeks she had had delusions of persecution and auditory hallucinations. She believed that the instigators were people who lived above her, who wished her room for a friend, and that the landlady was also involved. She called in a priest the night before to rid her room of devils that infected it. She wandered about during the night disturbing the house and did considerable screaming as though frightened.

On admission she was quiet, accessible, cooperative; approximately oriented as to place. She talked fairly clearly.

The history was obtained from a niece, who had heard from her mother that the patient had always been queer. She was constantly irritable, and after knowing persons a week or two would imagine that they were conniving things against her. She was never good natured or happy; she was melancholy and thought of unpleasant things. She was a malicious gossip, telling untruthful, slanderous tales about people she knew, saying that the women went with men immorally, etc. She had a vicious temper. Eight years ago she bent her sister's back across a railing almost injuring her. She was said to be very untruthful and jealous. She went to the theater and read love stories constantly. She was usually very haughty with men, but had had a mild love affair years ago. She had no friends and hated the world. This niece was the only person with whom she could get along, and of whom she was fond, although the patient also slandered the niece.

The patient had apparently led an uneventful life as a saleswoman until four years ago, when she was said to have given up work, hung around the house, worried continually about her physical condition, and saw many physicians. These found nothing wrong, except that she was anemic. Two years ago she had a peculiar spell of short duration and then became more queer than ever; she neglected her house and lost interest in everything. She would sit shaking her head and thinking for a long time; began to talk in a slow, drawling way, and became pale and thin. Often she would not answer questions, and would laugh in a silly way over things that were serious. She said that people were calling her names. She showed no fear, and gave no explanation for the voices. She was naturally reticent and seldom explained her thoughts. She said the people upstairs were peeping down on her, that men were going in her window and were under her bed. She continued to gossip and make trouble, and would write letters to do so. She once threatened her aunt, saying she would shoot her.

For about three weeks the patient had been eating very little and probably not sleeping much. She had become very thin. A day or so before admission she said a 21 year old boy who lived in the same house that she did had promised to marry her. She ran after him, so that he refused to come back to the house.

There was no history of fainting attacks, convulsions or shocks. Memory was unimpaired. No dizzy spells or headaches; no periods of confusion had been experienced. For two years she had thought that men looked at her on the street because she was so good-looking. It was said that shortly before admission she talked a great deal about the devil; ran up and down the steps with no clothes on, and did a lot of silly talking. A doctor was called and she was brought to the Boston State Hospital.

Examination.—The patient denied the majority of the statements given in the outside history, and those she admitted she explained away. She was sure that she had been quite nervous recently, and that this began from sheer hunger because she had no money. She denied the love affair with the boy. She said that she had commented on his good manner and on his studious habits. She told of a love affair at 22, with a man who deceived and wronged her, and then deserted her. It was not possible to get any slanderous remarks from her about any of the neighbors. She admitted that she heard the people upstairs talking about her, saying they wanted to get her out, calling her names, stamping on the floor so as to make it uncomfortable for her; also that she called the priest to bless her room, thinking it might be devils, but before she was discharged from the hospital she insisted that she was sure there were no devils there. She was at all times quiet, pleasant and agreeable, accessible, courteous. She gave no evidence of hallucinations while in the hospital, and no further evidence of delusion formation.

She was pale. The pupils were thought to react less well to light than to distance, but they apparently reacted normally to both. The heart was enlarged on the left side, and the sounds were weak. The pulse was bigeminal; blood pressure: systolic, 105; diastolic, 70; hemoglobin, 90 per cent. The differential count showed 5 per cent. of eosinophils, otherwise it was negative. Urine findings were negative. Spinal fluid globulin 1, albumin 1, cells 2, gold 0000111000.

It is to be noted that in this case there are two conflicting points of view. First, there is the view of the patient's relatives that she has always been queer, and that for four years there has been a distinct change in character, even more marked during the last two years; and, according to the account of the people where she lived, a definite exacerbation during the three weeks preceding admission, with a crisis the night before. A history of this type is, of course, not uncommon in the paranoid psychoses. On the other hand, there is the patient's denial of practically everything attributed to her, although it must be noted that her explanation for the things she admits is practically always inadequate. The acute episode could equally well be explained on the basis of exhaustion from prolonged undernourishment and anemia. The spinal fluid findings were not marked, but they were definite. Of course, protein increase in the spinal fluid occurs in a wide variety of conditions in which there is some disintegration of nervous tissues. The majority of such cases at this age are syphilitic, neoplastic or vascular in origin. In this case there was no evidence of any one of these three causes, aside from the spinal fluid changes themselves. With the Wassermann reactions on the spinal fluid and blood negative, little stress would be laid on these changes, were it not for the fact that occasional cases are seen in which findings of this type are the first indication of neurosyphilis, the spinal fluid findings later becoming more definitely positive and the Wassermann reaction showing positive instead of negative results.

GROUP 2. CYCLOTHYMIC CASES

CASE 7 (File number 9391).—*Man, aged 42, physical and serologic signs of neurosyphilis, psychosis of manic depressive type. Recovery from psychosis.*

History.—This man entered the hospital Sept. 16, 1917. The family history was negative, except for one sister who had had, about 1907, an attack of depression in which she had hallucinations, had ideas of persecution and poisoning and refused to eat. She was at the Worcester State Hospital two years, and following discharge recovery seems to have been complete.

The patient had a normal childhood. He became a lithographer and for twenty years had conducted a prosperous business of his own. He was married in 1907. His wife died following the birth of the first child. He had never been sick. About seventeen years ago he had had a sore on the penis. He had not been a drinker. He had suffered somewhat from a chronic intestinal disorder, and about two months before admission to the Boston State Hospital he went to a general hospital, where, during the routine examination, his blood Wassermann reaction was found to be positive. About three weeks before admission the patient became very much depressed. He kept repeating that he was gone, was no use to the world, etc. He accused himself of being sinful, and said that other members of the family would be lost because of what he had done, that he had disgraced the family by sexual offenses. He was sent to a sanatorium, gradually grew worse and became so markedly agitated that he was removed to the psychopathic hospital.

Examination.—Here, he was clearly conscious, oriented and his memory good. He was markedly self accusatory, and feared that he was to be executed because of his misconduct. Attention was maintained with difficulty. Flow of thought was very slow, and there was a good deal of retardation. He was much depressed, anxious and agitated.

On physical examination, the heart and lungs were found to be normal. The pupils were slightly irregular; the left reacted sluggishly, the right was stiff to light. Knee jerks and ankle jerks were not obtained. There was paresthesia of left leg; fine tremor of the tongue and fingers, and a suggestion of ataxia in his gait. The spinal fluid showed globulin, 3, albumin 3, cells 9, gold 5555533331. The Wassermann reaction on the serum and spinal fluid was positive. He was put under intensive treatment. He continued to be depressed and agitated, moaning and groaning. He was markedly retarded. In July, 1918, he was transferred to the hospital department, and since has been released on visit in a much improved condition. Since his release he had been doing some painting and has improved markedly so far as the mental symptoms are concerned.

It will be noted that he showed physical signs of syphilis of the nervous system, but none of the cardinal mental signs in the way of memory defect, loss of reasoning power, of calculating ability, etc. It was rather a fairly typical picture of a mixed manic depressive phase. Presumably in time the patient will develop further symptoms of paresis, but for the present it is not possible to say more than that he showed, in the presence of some physical and laboratory signs of syphilis of the nervous system, a psychosis of distinctly nonrelated type.

CASE 8 (File number 12482).—*Acute maniacal outbreak in a German naval officer with confusion and disorientation. No neurologic signs of paresis aside from slight tremor; good memory; grandiose and euphoric in the most excited stage; recovery from the outbreak in about eight days. Serology of neurosyphilis.*

History.—The patient was a German officer assigned to a ship transferred to the United States for use as a troop ship. Enroute to Boston from Europe, he became violent, attempted to stop the ship so that he could take a swim, and started a fire in his cabin which threatened to burn up the ship. He was so violent that he was unmanageable at the Naval Hospital and was admitted here in irons, talking, gesticulating, spitting in the attendant's face, and struggling to get free. He was disoriented, incoherent, spoke poor English in a harsh voice and was markedly active. During the first three days he was noncooperative, excited, talkative, incoherent and rather playful. In this phase he gave his name, age and address correctly, and varied his statement of where he was according to his immediate environment. When in the prolonged baths he said that he was in a submarine. He quieted down rapidly and on the fourth day he was very friendly, was oriented, very observing, would mimic the other patients, was very gracious in his manner, quite talkative and humoristic. Within eight days of admission he seemed to be in a quite normal condition. He gave a good history of himself and of his past. Retention of school knowledge was very good. He could give a good account of the war. He was somewhat alcoholic, but not more so than is usual.

At the age of 17 he had a genital lesion for which he received no treatment. In 1915 he had another genital lesion which lasted three weeks. On this occasion he was treated by a syphilographer at the naval hospital in Kiel. He was given four courses of treatment, each consisting of six intravenous injections of arsphenamin and six intramuscular injections of mercury. He received no further treatment. He stated that he was all right when he left Brest. His trouble arose when about three days out. He believed that the chief officer, under whose direction he was on the boat, did not understand him and so made some trouble for him and reprimanded him on two or three occasions. (It is possible he was somewhat too active and this was the cause of the reprimand.) He could not give a clear story of all the events that happened. He denied attempting to stop the boat and to go swimming. He explained the fire incident by saying that he had cut an electric wire in the cabin in order to light a cigaret, as he did not have any matches, and by rubbing the two cut ends together he made sparks with which he lighted a piece of paper and in that way lighted his cigaret, and in this way, also, the small fire started. At this time he seemed to have no particular delusions, although a day or so later he stated that he was going to return to Germany and purchase a boat with which he would make considerable sums of money. The boat could be bought for perhaps 200,000 marks. He had no money himself, but he was sure that because of his ability he could borrow the money from bankers. He realized that he had had an attack of mental trouble, and probably at the time of discharge was still slightly euphoric. There was at no time any emotional instability. At first his attention was quite distractible, but later it became normal. Where he had shown flight of ideas in his more violent phase, his ideation became of normal range, as did his motor activity.

Examination.—Physically he showed numerous small scars from acne all over the body, a slight tremor of tongue and extended fingers, a slight swaying in Romberg's position. The knee jerks were equal and within normal range. The pupils reacted to light and distance, and were described differently at different times. They were equal, were said at one time to react sluggishly to light, the right better than the left; consensual reaction was present in both; they both reacted to accommodation. He seemed to have no speech defect on either English or German test phrases. The physical examination was otherwise negative. The Wassermann reaction on the serum was positive; on the spinal fluid, at the first test, it was negative, but the second test was positive. The spinal fluid showed globulin 1, albumin 1, 255 cells, including small and large lymphocytes, endothelial cells, polymorphonuclear and plasma cells, a gold reading 5555543330. A second spinal fluid showed more intense reactions in the protein test, 248 cells, and a gold reading essentially the same as before.

This case presents an acute maniacal outbreak characterized by violence and clouding of consciousness, together with many of the cardinal signs of mania, but coming on more rapidly and clearing up much more rapidly after a shorter duration of attack than is true for a manic depressive psychosis. There were no cardinal signs of general paresis in any of the phases. The gold reaction, of course, was typically paretic. The high cell count is of equivocal value, but it is usually considered to point more in the direction of a cerebrospinal syphilis of the meningeal type than in the direction of general paresis. It is difficult, therefore, to say whether we were dealing with a spontaneous remission of remarkable type in a case of a general paresis, with an acute delirium associated chiefly with a meningeal type of neurosyphilis, or, indeed, with an association of an atypical sort of mania with neurosyphilis. In any case, it is probable that the causative factor was the neurosyphilis, and that treatment had to be directed toward clearing this up. The prognosis in the meningeal types is much more favorable than in the paretic type.

CASE 9 (File number 12862).—*Acute maniacal attack with hallucinations and delusions in a man, aged 43. No clinical evidence of neurosyphilis. Serology positive. Rapid recovery.*

History.—A Portuguese was brought to the hospital by the police on July 2, 1919, with the statement that he had been normal until the day before, when he became talkative, flighty and incoherent. He appealed to God, got down on his knees and crossed himself.

When seen in the ward, he was oriented, talkative, active; consciousness was entirely clear. He dwelt on a back injury, the fact that he was dirty, that God was going to aid him. God neither spoke to him nor put thoughts in his head. No ideas of grandeur were elicited, although he was euphoric. He denied that there was any trouble with his mind. He was easily managed and cooperated willingly.

Mental examination revealed no memory defect. The family history, as he gave it and as it was given by his brother, was negative. His personal history agreed with the history given by his brother. He had always been quite conscientious and obstinate. About two years before admission he injured his back. Following that he was greatly worried by the pain in his back, his inability to work, and the difficulties he had in endeavoring to secure compensation.

There had never been any mental symptoms until on the night of June 30, when he entered his cousin's bedroom about 11 o'clock, saying "I want you to go to church with me tomorrow at 10 o'clock to prove I am a true man of God." He was put to bed, but an hour later he partially dressed and ran into the street, crying "Mother, Father," then dropped to his knees, and began to pray and talked incoherently. He was brought into the house and to his room. He immediately upset all of the furniture, and began to mop the floor and wash the windows. During the rest of the night he was constantly in motion, repeating, "Man has deceived me, I believe only in God." He continued this way all next day, constantly active and restless. He could be persuaded to lie down, but would immediately jump up. On the morning of July 2 he locked himself in the bathroom and refused to open the door unless his cousin would knock on it three times and call his name three times. About six months prior to this he had complained to his cousin that he had lost all sexual power and desire. This was the only prodromal symptom obtained.

Examination.—Physical examination was practically negative. There was no evidence of any disturbance of nerve functions, so that whatever the injury to his back may have been it had not resulted in any definite organic lesion. The reflexes were all normal. The pupils were equal, circular, eccentric in position and reacted promptly to light and distance. Physical examination was otherwise negative, including the sacro-iliac joints. The urine was normal.

Mental examination revealed that he appeared to tire easily, and at times would not make the mental effort necessary to fix the date of an occurrence. At times he would become a trifle confused and speak about "God not wanting to kill anybody." He was completely oriented. He was continually interspersing his answers to questions with irrelevant remarks, such as, "Believe only in one God, that is all." Some of his remarks indicated the possibility of ideas of persecution, as "I cry all day and all night to go out with the good people. I appreciate, try to mind all people, but some people try to make bad out of you." When asked if any one had been trying to hurt him, he replied, "My feelings cannot be hurt out of any one. If you punch in my jaw two or three times, that's your chance, I don't care."

He gave a good and coherent account of his past life with the exception of such remarks as were interjected. He gave a history of sexual promiscuity until after his accident. He found that erections produced so much pain in his back that intercourse was impossible. He gave a good history of the accident, the compensation and his difficulties following that. There was no insight into his mental state. He admitted auditory hallucinations and some visual hallucinations. At times he was somewhat agitated, but, on the whole, he was rather indifferent. His activity was fairly normal while here, although there was a definite history of a pressure of activity. His mental condition improved markedly, so that by the fifth day he seemed entirely clear. He remembered the things he had done, but he could not account for them and was not certain of all of the details. He said, "It came so strong on my head it pulled me up and down the street, pull me this way, that way, make my head so big I feel like I go crazy." He did not speak of God, or "being a good man to God any more."

Because of his marked improvement, he was discharged to the family instead of being committed.

The serologic examination showed Wassermann reaction positive in blood and spinal fluid; globulin 1; albumin 1; gold 1134400000; 166 cells.

We have here to do with an acute maniacal outbreak, showing some hallucinations and possibly delusions of a religious nature, associated with syphilis of the nervous system, with quite rapid improvement under hospital care. Further outbreaks probably of the same nature are to be anticipated. The duration of the neurosyphilis is questionable, but certainly it must antedate by a considerable period this outbreak.

CASE 10 (File numbers 7724, 8514, 8563).—*Tabetic fireman, aged 38, with three attacks of grandiose mania, in which the paretic picture is typical. Intensive treatment; remissions with almost complete recovery, good insight and practical judgment, slight memory loss.*

History.—This patient was first admitted to the hospital Nov. 23, 1916, aged 38. He came voluntarily from a general hospital, following a suggestion that he take treatment in our syphilitic clinic. At this admission the patient was perfectly oriented. He said that his memory was not so good as it had been, although he gave a good, coherent account of his life and occupation. He gave a history of syphilitic infection in 1910, with treatment of mercurial inunctions for a period of two years. Other than that his past history had been essentially negative, except for some nervousness, irregular hours, and a good deal of exposure to the weather while attending fires. For about four years, namely, since 1912, he had had some difficulty in keeping his balance, and had had shooting pains in his legs. At this time he showed a little speech defect and sometimes repeated words. His school knowledge was well retained, and his calculating ability was good. His writing showed no tremor. Conduct was normal, and he cooperated well.

Examination.—Neurologic examination showed that the pupils were stiff; knee jerks and achilles jerks were absent; Romberg's sign was positive; the gait was markedly ataxic, and tests of the lower extremities showed poor coordination. The Wassermann reaction on the serum and on the spinal fluid was positive. The spinal fluid showed globulin 4, albumin 4, cells 41, gold 5555443100.

Course and Treatment.—He was discharged to the outpatient department and was under treatment there, and during this period worked, attending several large fires. About April 24, he became overactive, planning big schemes, involving fire protection for a large number of adjacent towns. He was restless, sleepless, talkative, quite euphoric. On admission he was perfectly oriented, repeated test phrases well, showed absent knee jerks and positive Romberg's sign. At this time a history of syphilis in 1889 was obtained, together with symptoms of tabes as long as ten years prior to admission, that is in 1907. These symptoms of tabes were given by the physician who had been treating him. He continued to be restless, very talkative, and had a large scheme for extending the service. There were pressure of speech and activity, some speech defect, but memory fairly well retained. Shortly thereafter the patient became very ill. He was restless, very talkative, confused, with generalized, marked tremor. He gave the month as the 4th of July, the day of the month as Friday the 16th, when it was neither. He said that he had had syphilis in 1819. He was now 38, born in 1917. The year was 1911. Asked how long he had been here he said "36 years of age." His speech was extremely disordered, and it was difficult to understand many of his words. He was put on the danger list, and it was felt that the end was rapidly approaching. However, he was given an intraventricular treatment and began to improve. He improved slowly, became quite rational and had insight into the attack through which he had passed. He realized that he had been out of his head. July 12, 1917, he was allowed to go on visit, returning June 17, 1918. He was euphoric, emotionally unstable, cried easily, with a vague feeling that he had been mistreated; he was quite excited, restless and circumstantial with a marked pressure of speech. He was rather grandiose, more or less disturbed, and continued so for about six weeks. He then began to quiet down and showed signs of deterioration, which have persisted to the present time, although there has been no further attack of excitement.

This case shows, under the influence of treatment, very remarkable remissions, resembling the remission of manic depressive psychosis, in which all symptoms except a slight deterioration disappear, and extremely good insight is attained.

Case 11 (File number 12861). — *Boy, aged 21. He had three attacks of maniacal type: serologic findings of probable early neurosyphilis.*

History.—This patient was sent to the hospital July 2, 1919, having been arrested in a neighboring city the night before when asleep on a bench. It was discovered that he had formerly been at this hospital, and at Taunton State Hospital, so he was returned to this hospital.

On admission he showed increased psychomotor activity, talkativeness, suspiciousness, and was somewhat resistive. In the ward he was accessible and cooperative, talkative, hyperactive, happy; quite circumstantial in his answers to questions. He was correctly oriented, and had a very good memory.

Examination.—Mental examination revealed some characteristics of mania, with the addition that he was quite critical and somewhat suspicious and slightly evasive. He was distractible, overactive, and showed a small range of ideas; he was elated and somewhat grandiose. No history of syphilis was obtained.

The physical examination was negative. The Wassermann reaction on serum was negative. The spinal fluid showed 14 cells, and a gold reading 1233100000.

Here is a patient with a history of two previous attacks of manic depressive psychosis, and at present slight alternations in the spinal fluid which are difficult to explain. It would be very important to know whether the positive fluid findings antedated his earlier attacks.

In the absence of any positive information, it becomes important to follow the case to determine whether eventually the attacks change in type.

GROUP 3. HYPOPHRENIC CASES

CASE 12 (File number 12802).—*A feebleminded woman, aged 30, without clinical signs of neurosyphilis; positive serology — congenital?*

History.—The patient, a hunch-back dwarf, was arrested for vagrancy and for sleeping on the Boston Common, which she says is "good for anyone." When asked why she was brought here, she said, "For drinking, I suppose." Why do you drink? "Why, because I like it, and when I like a thing I like it and I am not going to change no matter how many doctors is about. I have got a room and I don't bother nobody." She said a man was going to marry her; she had no other friend. She had one sister in the Boston State Hospital. Her father was said to be in the Long Island Hospital. Her sister was committed to the Boston State Hospital in October, 1918, with the diagnosis of dementia praecox, so that practically nothing could be learned about the patient's past, except what she gave herself.

Examination.—The patient was accessible, talkative, rather sullen at times, poorly oriented without marked impairment of memory, but unable to give details accurately, with no school knowledge, saying she was in the fourth reader when she left school. She had a very meager range of ideas; her fund of knowledge was extremely limited. The patient was markedly restless at times, quite circumstantial and talkative when once started.

There was a dorsal kyphosis with pigeon breast. The pupils reacted poorly to light, well to distance. Neurologic findings otherwise were negative. Wassermann reaction on the serum and spinal fluid positive; globulin 0; albumin 1; cells 20; gold 0012332100.

The mental level was 6.8. The patient was cooperative.

From the patient's account of her own life, her poor retention of school knowledge, her limited fund of general information, and her general temperament, it seems quite clear that the mental age represents the highest the patient has attained. In addition, there is neurosyphilis, the origin of which is distinctly obscure. She gave no history of syphilis and, in fact, denied sex intercourse. With the kyphosis, and the possibility of its being tuberculous, one would have to consider a low grade form of tuberculosis of the nervous system. The positive Wassermann reactions, however, would seem to rule that out, and it seems more likely that the kyphosis was due to a disordered bone metabolism in childhood, or to syphilis, rather than that the spinal fluid changes were due to tuberculosis.

In any event, this is a case of feeblemindedness with syphilis of the nervous system, demonstrated at the age of 30, which has possibly been existent since birth; in other words, possibly congenital syphilis without any of the customary signs of that disease.

CASE 13 (File number 13394).—*A feebleminded boy, aged 19, with a mental age of 7½. No clinical signs of congenital syphilis or of neurosyphilis, but moderately positive spinal fluid.*

History.—The patient was arrested on the charge of sodomy, which had existed for several months with a younger boy then out on visit from a school for the feebleminded. He was a typically feebleminded youngster, whose fund of general knowledge and reactions agreed closely with his mental age.

The family history was negative. There was no history indicating syphilitic infection of the parents. The patient had had diphtheria at the age of 3, and was very sick at that time. He had never had a head trauma. He had always been very quiet, and did not associate much with other boys. He was backward in school, reaching the fifth grade at the age of 15.

The patient considered himself normal, and was not much concerned over his present situation.

Examination.—The physical examination was entirey negative for signs of congenital syphilis, and for neurologic and mental signs of paresis. The blood Wassermann was negative. The spinal fluid showed albumin 1, globulin 1, 3 cells, and a gold reading of 0012110000.

These signs are equivocal for neurosyphilis, but do indicate an organic disease, which from experience we know is most likely to be a neurosyphilitic one. As previously pointed out, a negative Wassermann reaction in the blood serum is not sufficient evidence on which to exclude neurosyphilis, in case there are other positive tests in the spinal fluid, and this is particularly true in cases which may have a congenital origin. No other hypothesis will so satisfactorily account for the clinical and serologic picture.

GROUP 4. CASES WITHOUT PSYCHOTIC SYMPTOMS OR CLINICAL EVIDENCE OF NEUROSYPHILIS

CASE 14 (File number 10524).—*No nervous or mental symptoms of neurosyphilis. Constitutionally inferior girl, aged 20. Serology positive. Treatment. Doing well in community.*

History.—This is an extremely interesting case of nonsymptomatic neurosyphilis. The patient was admitted to the hospital April 11, 1918. Her father was reported to be a fighter, intemperate and abusive, and took no responsibilities for supporting his family. He died of tuberculosis in 1905. The mother worked out by the day, and was honest, hard working and moral. She was supposed to drink a little. The patient had four brothers, all wild. Two had court records for larceny. One was in the Lyman School, another was a conductor and supported the mother.

The patient left school when in the sixth grade, at the age of 14. She then worked in a candy factory, from which she was discharged for being disorderly. Later she was sent to the House of the Good Shepherd. She then worked irregularly, receiving no definite discipline in the home. The medical history was not especially remarkable. At the age of 14 she began to dress like a woman, painted and powdered, and often stayed out until midnight; on several occasions she stayed away from home over night. She was immoral with many different men, but denied receiving money. Later, under training and supervision to which she responded well, she began to work out, and did her work well under guidance. She would lie to defend herself. She showed no immoral tendencies, and returned to her home to live. While there she tried several positions, but left all without giving notice. She would stay out until morning hours, and was frequently away all night. She was obviously, judging from letters and other evidence, leading the life of a prostitute and was sent here for observation.

Examination.—While here, she was quiet, accessible, cooperative and talked freely. There were no hallucinations and delusions. She seemed to have a very poor moral conception, and manifested no particular concern over her immoralities.

The physical examination was negative. There was slight tremor of the extended hands, and the knee jerks were active, but the pupils were normal; speech was normal, and there were no signs to indicate any organic involvement of the nervous system. Her mental age was 13. However, the examination of the spinal fluid on April 13 showed globulin 1, albumin 1, cells 76, gold test 5443322100. Because of some doubt whether this could be true in the absence of any symptoms, the puncture was repeated on April 18. The results were: globulin 1, albumin 1, cells 132, gold 4443333221.

Course and Treatment.—The patient was then discharged to the outpatient department where she was under treatment, it having been found in the meantime that the Wassermann reaction on the blood serum and spinal fluid was positive. In August she returned for a lumbar puncture, this time showing globulin 12, albumin 12, cells 1, gold 4433221100.

In the meantime, the patient had been working quite steadily and continues so to this day. Whereas she had not been considerably disturbed about her immoralities, she was considerably disturbed when informed that she had syphilis and that the outcome might be bad, and has cooperated well under the treatment.

This, then, represents a type of case in which, in the absence of any mental or nervous symptoms, we find marked changes in the spinal fluid, indicating an active invasion of the cerebrospinal axis. This case is quite similar to one of those presented in my previous paper.

CASE 15 (File number 13295).—*A boy, aged 15, with conduct disorder. Not psychotic, nor feebleminded. Character change at 11. Spinal fluid findings equivocal, but probably indicate neurosyphilis, possibly of congenital origin.*

History.—This boy was sent in with the statement that he acted surly, struck his mother, refused to go to school, and was placed at work and refused to work. He acted stupid at times. The father thought his mind might be affected.

The family history obtained from the father was negative. The mother was described as a normal person. The father, himself, seemed normal. He was a traveling salesman; possibly had had syphilis. There was no miscarriage. The older sister was in a school for the feebleminded; the younger sister was said to be bright, active and sociable.

The boy was always considered bright. He graduated from the eighth grade at 15, having spent two years in the sixth grade, when he began to have difficulty. He lost interest in school, did not concentrate, and spent much time reading. He had always been somewhat surly, not responding well to correction or discipline. He was active, and played with boys of his own age. He never had had a fight.

The character change seemed to date back to about the age of 11. Of late he had been running away. He had seemed to be frightened when his father corrected him.

Examination.—The physical examination revealed nothing remarkable, except certain minor signs of malformation in the head. He was not sociable in the ward, and showed some slight mannerisms and squinting. He admitted his seclusive makeup, masturbation and smoking. He did not work well because the boys were too rough. He admitted having a bad temper, and was somewhat jealous of his sister. He seemed emotionally indifferent, not very attentive, quiet and kept by himself. His intellectual level was 12.4. He had no hallucinations, no delusions, no somnambulism, but had nocturnal enuresis until the past year. He seemed somewhat nervous, but showed no conduct disorder while in the hospital.

The Wassermann reaction on the blood serum and on the spinal fluid was negative. Other spinal fluid findings were: globulin 1, albumin 1, gold test 0023210000, twelve small lymphocytes per cubic millimeter. There was no history of trauma. There were no abnormal lung findings pointing to tuberculosis, and no evidence of any type of tuberculous involvement in the way of temperature, signs of meningitis, alterations in reflexes, etc., so that it appears most likely that the spinal fluid findings are due to a low grade syphilitic infection of the nervous system, probably congenital in origin. Incidentally, it is not particularly rare in cases of congenital syphilis in which the nervous system is chiefly involved, to find a negative Wassermann reaction and negative physical signs, with some alteration in the spinal fluid, which cannot be accounted for on the score of trauma, brain tumor, or tuberculous meningitis.

CASE 16 (File number 9282).—*Man, aged 40, without clinical evidence of neurosyphilis. No serologic evidence except Wassermann reaction positive in spinal fluid. Despite mild treatment, marked increase in serologic findings in two years, with no clinical symptoms.*

History.—This patient entered the hospital voluntarily for a lumbar puncture because his wife had developed paresis. At the time of this first examination the blood Wassermann reaction was negative in four different tests, but the spinal fluid gave a positive reaction in 1 c.c. dilution. The other tests on the spinal fluid could not be done as the fluid was bloody. He had, however, 9 cells. Because of this weakly positive Wassermann in the spinal fluid, and his wife's death from general paresis, the patient was kept in view and under mild treatment for a considerable period. The results of these tests are appended.

At no time has the patient shown any definite mental or nervous signs of invasion of his nervous system by the spirochete. His mental state has been one of some anxiety and apprehensiveness regarding the future, as he was very much impressed by his wife's death. He has continued to handle his business efficiently and, aside from insomnia and periods when he is more actively apprehensive, during three years he has continued to show no further signs.

Throughout the fall of 1917, 1918 and 1919, until the present, this patient's blood Wassermann reaction has continued to be persistently negative, although repeatedly tested. In January, 1918, the Wassermann reaction on the spinal fluid was again positive. No other tests were recorded. In November, 1918, lumbar puncture showed globulin 1, albumin 2, cells 25, a gold reading 5554443211 and a positive Wassermann reaction. As previously stated, however, the patient has not up to the present time displayed any mental or physical symptoms which we regard as characteristic of paresis, although the spinal fluid findings have become stronger.

Treatment.—In this case I should also like to point out the failure of what might be called moderate treatment to prevent further inroads on the nervous system. During November, 1917, the patient had three injections of 0.3 gm. of arsphenamin; during December, two. In 1918, for the entire year, there were twenty-two injections of diarsenol of 0.3 gm. each, and one injection of mercury. The latter made the patient very sick, so has not been attempted again. During 1919 the patient was given six intravenous injections, a total of thirty-four injections in a year and a half, and as previously shown, the spinal fluid tests now give more evidence than ever before of the invasion.

This case is important from several points of view. In the first place, it shows that a negative Wassermann reaction in the blood serum is not sufficient evidence of noninvasion of the nervous system by the spirochete. In the second place, it shows the long duration of time during which mild change may be present in the spinal fluid before any definite mental or neurologic symptoms are encountered. In the third place, it demonstrates the failure of ordinary methods of treatment in combating a progressive disorder, such as syphilis of the nervous system.

CASE 17 (File number 5970).—*Boy, aged 22, unstable type, liar, wanderer, inventor, economically inefficient. Regarded as not psychotic but serologic findings of a paretic process. Diagnosis uncertain.*

History.—This patient was admitted to the hospital Oct. 16, 1917, from the base hospital at one of the military camps. The statement received from the military camp indicated that he had had no previous attack and that the present attack had begun about October 10. He had had a previous head injury, not serious, and the legal papers gave the following statement: "He used to run away from school at 11 years, take a rifle and study nature in the woods, deer and muskrat. Fell from a hen house, when 6 years old, on head. Typhoid at 2. Shot by boy companion when hunting rabbits. Malaria in 1913. Gonorrhea four times." "I have talked with 100 men and ten officers and have accurate account of what each man thinks and have compared this with the same number of men in England, and have worked out the percent. I can furnish the U S. A. with meat at 4 cents a pound." "I can put my hands on millions; no one knows it, but I can." "I have proved I can introduce foodstuff." I have invented a kerosene carburetor."

At the time of admission, the patient stated that he had a great invention which would start an engine on kerosene. He had been working on it for two years, and was afraid people would steal it. He said he could supply the army with meat without beef. They thought he was crazy because he walked up and down, but he could easily prove to anybody that his inventions are of great value.

A detailed history was obtained from the family, which indicated that the patient came of a long lived family, in which no psychopathic determinants were found. He was one of eleven children, all of whom were apparently normal, except for the statement that many of the paternal relatives had a roving disposition.

The boy had less than a grammar school education, learning without difficulty. He was always very active and vivacious. Since about the age of 12 he had been disobedient and difficult to control. Sometimes he played truant from school. He never had much sense of responsibility, and was not very dependable. He was very popular with the boys, witty, quick tempered, not resentful and seldom depressed. He over-estimated himself and his abilities and assumed that every one shared his opinions. He had a general reputation of being untruthful; he told "tall" stories of his own wonderful experiences and achievements, such as the number of deer he had shot. He was very fond of hunting. He had a good deal of mechanical skill, but had not worked steadily. He was of a roving disposition. He left school because he had trouble with the teacher. He did some work in a garage, driving automobiles and repairing. The father tried to discipline the boy for the first time at 17, and he left home. For a year and a half he worked as a chauffeur in West Virginia; then returned home, and for two and a half years worked irregularly, usually in automobile shops. In 1916 he went to New York City, where he worked for an automobile concern. Later he called himself "Huppy" Smith, and claimed to be a great race driver. He returned four or five months later. He told his family that he had made one or two trips across the water, taking care of horses on a boat. They did receive some cards from him mailed at London and Liverpool. He worked for a time in an automobile shop, but was habitually late; then for a time with his father, and then as overseer of men in the repair of roads. His family regarded him as something of a mechanical genius, and had petted and praised him a great deal on that account.

At the age of 17 he had what was supposed to be a Neisser infection, which was treated for two or three months and is said to have been cured. He was hypochondriacal and made a big fuss at a little pain.

He was drafted into the army and two days later began to write to his people that he was not fit for army service A week after he went to camp the family visited him. They found that he had lain five days on his cot with his coat folded and placed under his head for a pillow. They were told that he had been inoculated for typhoid fever and directly after had fallen against a radiator and cut his head. By October 13 he was much disturbed because he had had no opportunity for bathing, was nervous and homesick. After his usual manner, he told exaggerated stories.

The informant, a brother, gave no history of hallucinations or of what appeared to him to be delusions. He thought that the patient's ideas in regard to his invention were not irrational, that he had been working on it for two years. There was no history given of convulsions, syncope or other physical symptoms.

Examination.—The physical examination showed that he was well developed and well nourished. The face was asymmetrical, the nasal septum deviated to the right; there was a mucopurulent discharge from the nose. Some teeth were missing; others carious. Heart and lungs were normal. The blood pressure was: systolic, 120; diastolic, 80. The pupils were equal and regular, and reacted to light and distance. Knee jerks were active and equal. There were no abnormal reflexes. The ophthalmoscopic examination was negative. Roentgenogram of the head gave no evidence of trauma. The urine was negative.

On mental examination it appeared that the patient was quite satisfied with himself and discussed his hopes quite freely. He was anxious for assistance to present his plans to the authorities. He seemed somewhat overactive, although not markedly so. He was extremely talkative with a remarkable flow of speech. He was correctly oriented, and did not consider himself ill but in a dilemma. He appeared to believe that because of his deliberate intention to make himself nervous and ill he was sent here. He asserted that he wanted to appear queer so that he might get his plans before high authorities. He also stated that he had said all the queer things he could think of when brought before the doctor. His memory was quite good, but in his account of his past experiences he showed an egotistic attitude. He stated that at 15 he started to see the country because he felt that he had had little education. He would work a few days, then travel to another place. He named a great group of towns in West Virginia in which he claimed to have lived. He said he had been in New York, New Jersey, Kentucky, Tennessee, Ohio and Arizona, and that he had traveled over nearly every state in the West. He said that he had falsified his age when 16, in order to vote. He gave a long and circumstantial account of his wanderings over the United States and crossing to England, and a very detailed account of all the things he did while working in the garage at home. He asserted that following a fire in a large private game preserve, he had noticed the next year that the deer were feeding on the tender shoots in the burned district, and yet the bushes and trees and grass grew and became more luxuriant than on similar land outside the preserve. So the idea came to him that deer feeding on vegetation did not destroy it, but apparently did it good, and, therefore, a given number of deer per acre would be self-sustaining having always a constant source of food supply. On this problem he had been working ever since. He says he has been a mighty hunter, having killed many deer, both in and out of season. He finally calculated that the acreage required for the deer was the same as for the cow in pasture, one half acre per head. He gave elaborate details on the subject of the care of a herd, breeding, etc. He worked out two schemes, quite absurd, for feeding the army meat without using beef.

The patient also gave a long, detailed mechanical description of a carburetor which would use kerosene instead of gasoline.

On the basis of the history that the boy had always been queer, a good deal of a liar, a rover, and that ideas of the type presented above had been frequent, together with the family's feeling that he was a mechanical genius, we regarded him as not psychotic. He was overactive and quite talkative, and emotionally elated and egotistic. But as all of these things were described as being a part of his usual makeup, and as his intelligence was quite up to that of the average adult, we conceived that he was a case of psychopathic personality. The Wassermann reaction on the serum, however, proved to be positive, whereupon a lumbar puncture was done, which showed globulin 1, albumin 1, cells 63, and Wassermann reaction strongly positive. Another neurologic examination proved to be negative. Further inquiry did not reveal a memory defect or any of the characteristic or essential symptoms of general paresis. Accordingly, the classification of the case is extremely difficult, although it is obvious that we have to do with a case of neurosyphilis, whether congenital or acquired is not certain, in a boy who otherwise shows very little except psychopathy. Of course, the ideas which he presents are absurd, but not more absurd than the beliefs of many people who are regarded as normal. It seems clear from the facts obtained that if neurosyphilis did not exist from the first, we deal with a case in which developing neurosyphilis has not so far altered the clinical symptomatology.

CASE 18 (File number 12051).—*Pseudoparesis in a man, aged 33. No syphilitic history. All serology negative.*

History.—This case is presented from an entirely different point of view. The man was first seen in consultation at a general hospital, where his memory defect, euphoria, general dulness, tremor, speech defect, loss of calculation ability, pupils and incontinence led to a diagnosis of general paresis, but the blood and spinal fluid findings were negative. Because of this the patient was transferred from the general hospital to our institution on Jan. 28, 1919. For the past few months the patient had become forgetful and it had become difficult for him to think and focus his attention. He was very drowsy a great deal of the time and had headaches and pains in his legs. On admission it was difficult to hold his attention and very little information could be obtained because of his poor memory. He showed a speech defect. He gave a history of influenza in November, with good recovery.

Examination.—On physical examination nothing abnormal was found on the sensory side. His speech was defective. Test phrases were repeated poorly. The gait was wide based and shuffling. Station was not defective. He had coarse tremor of the hands. The superficial reflexes were all present; the deep reflexes were present and active. There were no abnormal reflexes. The pupils were slightly irregular, but reacted to light and distance. The patient was incontinent.

Mental examination revealed approximately what has been given before; namely, that he was somewhat euphoric, and attempted to cooperate. His memory was very poor, especially for recent events. Retention of school knowledge was poor. He could not give a good history of his own past life. He gave a past history of malaria in 1906 and in 1910. He had been alcoholic when in the army, but could give no additional facts. There were no delusions and no hallucinations. Associational range was narrow. He had complained of severe frontal headache, slight blurring in vision, projectile vomiting, but no attacks. All the time he had been under observation, either at the general hospital or here, his blood was negative to the Wassermann test, and his spinal fluid negative to all tests. We considered the case as probably one of frontal lobe tumor for reasons that need not be given here.

My sole purpose in reporting this case is to raise anew the question of pseudoparesis, which is to my mind of doubtful value as a conception. The case also brings forward the point that neurologic signs, nonfocal in character, of generalized cerebral invasion, may be produced by other things than the spirochete, and that, accordingly, clinical evidence alone is not enough to establish the presence or absence of spirochetes in the brain. This case has wide and important bearings on the question of the postinfluenzal psychoses, and will later be considered in that connection.

Case 19 (File number 12244).—*A man, aged 36, with a hemiplegic stroke at 34; neurologic signs of organic disease; serology negative. Treatment; two months later serology positive.*

History.—The patient was admitted to the hospital on voluntary application, March 6, 1919. He complained of inability to concentrate, following a stroke in October, 1917, and seemed to be mildly depressed. The mother seemed to have been somewhat peculiar, having attacks of hysterics, so-called, at the time of the menopause.

The patient was a high school graduate. Later he was successful in the wool business, going into a larger concern on a partnership basis in October, 1917. He was ousted from the firm and there had since been a great deal of difficulty about arranging financial matters in connection with his interest. His wife regarded him as an affectionate, sensitive man, nervous because it was difficult for him to keep still. He did not worry, did not care for dances or the theater, was bright, well informed and fond of outdoor sports. He had some impediment in his speech. He was not a drinking man until the time of his business difficulty, when he began drinking, the exact amount was unknown.

Oct. 23, 1917, he had had a stroke. He had previously complained of feeling tired and found it an effort to do his work. At the time of the stroke his face was drawn to the left; his speech was thick; he said there seemed to be a film over his left eye. His right arm and leg were paralyzed. He fell, losing control of his limbs, but regained the use of his leg the following day. He remained in bed two weeks, and at the end of that time was able to walk. He was unable to cut meat for himself until December, and until that time it was difficult for him to keep food in his mouth. From December to April he was in a sanatorium; he spent his time in outdoor sports. When he returned to Boston he felt well enough to go back to business, but was unable to start anything because of the situation then existing in the wool market. He made an attempt to do other kinds of work, but this depressed him, and he felt unable to do so. In July he became very much depressed and somewhat later disappeared. He wrote letters that he was going to the Maine woods, then that he was going to Chicago. Ten days later he was in Boston. He said that he had been in a hospital in Chicago, but could not remember its name or why he had gone there. He then seemed somewhat better, but did not return to work. He had a slight attack of influenza in September, apparently without bad results. In January, 1919, he again became depressed. He refused to mingle with people. Once he said, "You know what I am going to do if my head doesn't get better. I have often thought of killing myself," but he made no attempt at suicide. He complained that some of his thinking power had been taken away. He seemed to lack attention and concentration.

Examination.—Physical examination revealed that he was well developed and well nourished. There was impaired vision in the left eye. There was no disturbance of ocular movements; no abnormal reflexes. Gait and station were normal. There was slight tremor of the tongue. All superficial and deep reflexes were present. Knee jerks were unequal, hyperactive. Pupils were dilated and reacted to light through a narrow range. The blood pressure was: systolic, 120; diastolic, 85. The spinal fluid was entirely negative. The Wassermann reaction on serum and spinal fluid was negative. The left eye-ground showed a peculiar arrangement of vessels in the disk, and probably some slight choking. The right eye-ground was negative.

The patient was easily disconcerted if questions were asked too rapidly, but his memory was good and he gave a coherent and detailed account of his past life. He seemed somewhat depressed. He was slow to answer and used stereotyped expressions.

A very careful and thorough neurologic examination failed to reveal anything indicating a focal lesion. A total of seven blood Wassermann tests were negative, both before and after provocative arsphenamin injection. A roentgen-ray examination was practically negative.

The patient was pleasant and cooperative and gradually became somewhat less depressed. He then went voluntarily to the Grafton State Hospital, where in May, 1919, another lumbar puncture was done, showing a positive Wassermann reaction, albumin normal, globulin 0, cells 57, gold test reading 2442330000. The Wassermann reaction on the serum was negative.

Accordingly, we have here a case with a definite history of a hemiplegic stroke occurring eighteen months before the patient was seen, with considerable recovery of function, but with some difficulty in concentration, attention and rapidity of thinking and acting. No history of syphilis was obtained. He had been somewhat alcoholic immediately prior to the stroke and was, at the time of our examination, not psychotic, although showing some neurologic findings. The negative serology would seem to exclude neurosyphilis, but under the influence of treatment the spinal fluid showed markedly positive findings in the way of Wassermann reaction, cell count and gold reaction, although the Wassermann test on the serum and the protein content remained negative. Hence, in the presence of neurologic signs, a negative serology is not enough to exclude neurosyphilis. Accordingly, the importance of equivocal signs is emphasized.

SUMMARY

1. Nineteen cases are presented, of which fourteen are undoubted cases of neurosyphilis, one a case of pseudoparesis and four have negative physical and equivocal serologic findings.

2. Of the fourteen undoubted cases, only four present clinical evidence from which a diagnosis of neurosyphilis could be made.

3. Neurosyphilis occurs more frequently in association with paranoid psychoses than is usually recognized. Although six cases are here reported, not one is a clear example of the syphilitic paranoid psychosis, two being heavily alcoholic, one a paretic, and two possibly not neurosyphilitic, though showing changes in the spinal fluid.

4. Any type of abnormal mental state, or no abnormal state, may occur with neurosyphilis. It does not follow that all such cases are paretic.

5. Serologic findings may definitely increase under treatment, with or without increase of clinical signs, emphasizing the importance of provocative treatment in rare cases.

6. A negative blood Wassermann reaction, or many such, are not always conclusive negation of the presence of neurosyphilis.

7. A negative spinal fluid is not absolutely conclusive evidence of the absence of neurosyphilis.

8. Since neurosyphilis may exist in association with any type of mental symptoms, and since such states may exist in the absence of any of the usual signs and symptoms of neurosyphilis, it follows that lumbar puncture should be done at least in all cases which present any atypical features. It is equally important to puncture cases with any clinical signs of neurosyphilis, since the signs may be misleading.

9. The prognosis and treatment of any cases are always modified by the presence of neurosyphilis.

10. Neurosyphilis as a disease may exist for a long period before symptoms appear.

HEREDITY IN EXOPHTHALMIC GOITER

WITH A REPORT OF TWO JUVENILE CASES *

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In his excellent work on heredity, Dejerine[1] pointed out the fact that exophthalmic goiter occurs in neuropathic families. He looks on exophthalmic goiter as a form of an hereditary neurosis. Long before Dejerine, Rendu[2] wrote that persons with this disease had among their ancestors, aliens, epileptics and general paretics. Rendu claimed that there must be a certain relation among all these diseases. Since then Gauthier[3] has shown the important rôle played by the thyroid gland in Basedow's disease. Several writers on the subject have attempted to show that there is a direct heredity in exophthalmic goiter. Pende[4] states that primary exophthalmic goiter is always associated with a constitutional hereditary state of irritability and instability of the thyroid, or the regulating nervous system of the thyroid secretion. Pende bases his statements particularly on the reported cases of hereditary exophthalmic goiter. It must, however, be admitted that a fairly careful search of the literature on the subject fails to show an abundance of direct heredity in this disease. Frey[5] reports a family of four, a father, aged 46, two daughters and one son, all suffering from exophthalmic goiter. Schultheiss[6] quotes a number of instances from the literature and reports on a family, several members of which suffered from exophthalmic goiter, as well as from diabetes and epilepsy. Leopold-Levi[7] in discussing the rôle of the thyroid instability. Carder in discussing Levi's cases states that the mendelian law of heredity may be applied to exophthalmic goiter disease. Baillich [8] reports a case of a girl, aged 6½ years, suffering from exophthalmos;

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* Read before Neurological Section, Academy of Medicine, November, 1919.

[1] Dejerine: L'heredite, dans les maladies des systeme nervaux, These d'agrey 1886, p. 157.

[2] Rendu: Quoted from E. S. Schulmann: Le goitre exophthalmique syphilitique, These Paris, 1918.

[3] Gauthier, de Charolles: Quoted from Longuez Lermagez: Rev. neurol. **26**:20, 1919.

[4] Pende, N.: Endocrinologia, Milan, 1916, p. 782.

[5] Frey, E.: Jahresbericht Neurologie und Psychiatrie **13**:753, 1909.

[6] Schultheiss, E.: Ueber Erblichkeit Morbus Basedow, Inaugural Dissertation, Jena, 1909; Ztschr. f. Neurol. und Psychiat. **13**:750, 1909.

[7] Leopold-Levi: Familles thyroid et dysendorm, Gaz. d. hôp. **86**:381, 1913.

[8] Baillich, G.: Goitre exophthalmique chez une fillette des sept ans heredite similaire, Bull. et mém. Soc. méd. d. hôp., de Par. **30**:708, 1914.

her father suffered from exophthalmic goiter, her paternal grandmother had a goiter and cardiac trouble, and her mother was extremely nervous. This author believes the disease is transmitted through females. Chapin[9] discusses the subject of transmission; contrary to the established belief this author proves that women suffering from exophthalmic goiter are prone to great fecundity. His statistics on forty-four women gave to each an average of 4.3 children while that of the normal is 2.5. Many of these women were below 30 years of age. Pregnancy is not affected by exophthalmic goiter and miscarriages are rare. Chapin further shows that the mortality of these children is rather high and that heredity presents itself in similar conditions and other nervous heredity.

Dubrenil-Chambardel[10] also reports a high mortality in infants of mothers suffering from exophthalmic goiter. He further reports a family in which an hysterical mother gave birth to ten children, eight of whom suffered from Basedow's disease. Cases of similar and dissimilar heredity are also cited by Bayloe.[11] Gauches and Salin[12] report a case where hereditary syphilis showed itself in the form of polyglandular hyperthyroidism. Schulmann[13] goes rather more deeply into the subject. After reviewing the literature and citing his own cases, Schulmann comes to the conclusion that hereditary syphilis plays an important part in disturbance of thyroid secretion, and in certain cases a more or less typical form of exophthalmic goiter may result.

Lonques and Lermoyez[14] report a family with seven cases of exophthalmic goiter. Some further reference to the literature may be found in that article.

REPORT OF CASES

Case 1. *History.*—M. B. (Fig. 1), aged 10, came to the Mount Sinai dispensary in September, 1919, with the complaint of swelling in the neck of three months' duration and "nervous spells." Physical examination showed that the child had a pulse of 156; blood pressure—systolic, 125; and diastolic, 85; marked exophthalmos and a marked Möbius' sign. There was a fine tremor of both hands; the face was flushed; there was considerable emaciation; the pupils were markedly dilated and the boy was in a state of apprehension and excitement. There was a marked increase in cardiac dullness to the left, dermagraphia was present and all the reflexes were exaggerated. Urine was negative; hemoglobin, 70 per cent.; red blood cells 4,024,000; white blood cells 7,200; polynuclears, 62 per cent.; small mononuclears, 29 per cent.; large mononuclears, 7 per cent.; and eosinophils, 1 per cent. Roentgenographic examination the next day showed marked enlargement of the thoracic aorta together with an exaggerated cardiac action. There was an indistinct shadow in the anterior mediastinum which surmounted the aorta and was somewhat suggestive of an enlarged thymus.

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[9] Chapin, A.: De la descendance des Basedowiennes, These Paris, 1910.

[10] Dubrenil-Chambardel: Un cas des Basedow hereditaire avec scoliose hereditaire. Province méd. 1907, p. 262.

[11] Bayloe: Arch. Med. Tolouse 1896, p. 30.

[12] Gaucher et Salin: Insuffisance ovarienne et syndrome des Basedow fruste? Une heredo-syphilitique, Bull. Soc. franç. de dermat. et syph. **23**:62, 1912.

[13] Schulmann, E. S.: Le tique goitre exophthalmique syphilitique, These Paris, 1918.

[14] Lonquez et Lermoyez: Rev. Neurol. **26**:20, 1919.

While examining the boy, it was noticed that his mother (Fig. 2) who accompanied him presented a typical picture of Basedow's disease, and inquiry elicited the following data.

Case 2.—*History.*—Mrs. B. C. (Fig. 2), the mother of Case 1, is 29 years of age. Her family history shows that her father died from diabetes and her mother suffered in earlier life from attacks of cardiac palpitation, had an enlarged thyroid and had lost considerable weight; but as she grew older her symptoms improved and the swelling in the neck diminished. One sister of the patient underwent operation for exophthalmic goiter at the Mount Sinai Hospital, and another sister is also suffering from a goiter. Mrs. B. C. has had three children and no miscarriages. Only one child is healthy, the third child, a little child aged 6, is suffering from an enlarged thyroid gland. This case will be reported below.

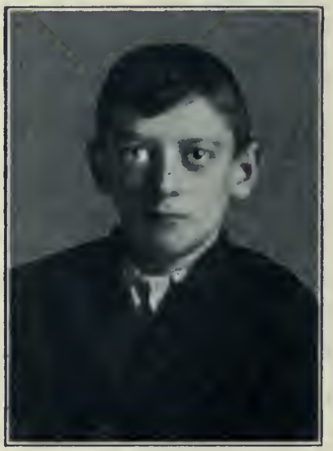

Figure 1

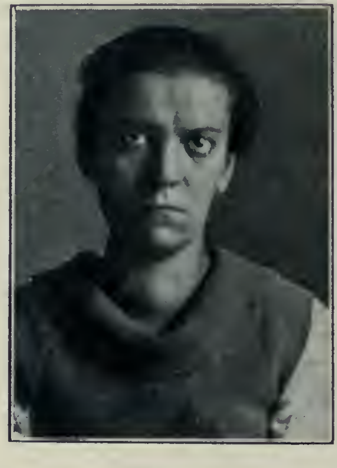

Figure 2

Fig. 1.—Exophthalmic goiter in a boy aged 10.

Fig. 2 (Case 2).—Mother of patient in Case 1, suffering from exophthalmic goiter.

Examination.—The patient stated that she suffered from frequent cardiac palpitation similar to that of her mother, profuse perspiration, attacks of diarrhea, restlessness and insomnia. There was a marked exophthalmos, and a marked Möbius sign. The patient was emaciated, had a fine tremor of the hands and a tremor of the head was distinctly felt when the examiner's hand was placed on top of it. She had a markedly enlarged thyroid particularly of the left lobe, and marked dermographia. The reflexes were lively. The pulse was 140; systolic blood pressure, 138; and diastolic, 85. She had marked pyorrhea. The urine was negative; hemoglobin, 60 per cent.; red blood cells, 3,728,000; white blood cells, 8,000; polynuclears, 52 per cent.; small mononuclears, 37 per cent.; large mononuclears, 10 per cent., and eosinophils, 1 per cent. Roentgenographic examination of the chest showed marked over-activity of the heart with distinct dilatation, hypertrophy of the left ventricle, considerable dilatation of the aorta, but no evidence of an enlarged thymus or substernal thyroid.

At the request of the examiner, the mother of this patient and grandmother of the first patient was brought to the clinic.

Case 3.—*History.*—Mrs. M. W., aged 55, a widow, whose father lived to old age, and whose mother was a very stout woman, had three daughters and one niece who suffered from exophthalmic goiter, and one of her sons was insane. Her husband died from diabetes. The patient was well until about fifteen years before she presented herself when she began to suffer from cardiac palpitation and to lose weight rapidly. About one year later she suffered from flushes, and at this time the menopause set in. Her cardiac palpitation continued until about two years ago. At the time of the onset of the thyroid symptoms the gland began to swell, but later on this subsided. After an intra-abdominal operation three years ago, the thyroid symptoms apparently disappeared. At presentation the patient complained of general weakness and rapid exhaustion.

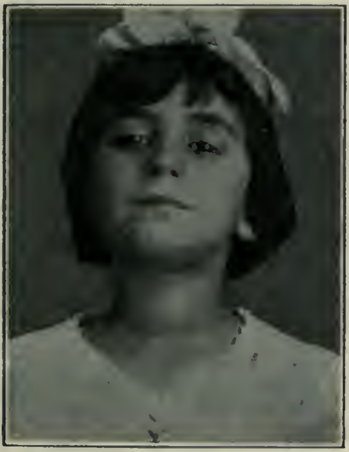

Figure 3

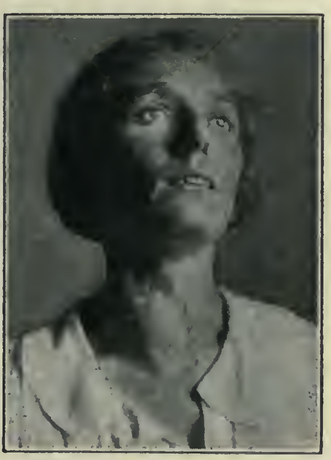

Figure 4

Fig. 3 (Case 4).—Child, aged 6, with exophthalmic goiter.

Fig. 4.—Woman, aged 33, with exophthalmic goiter. Maternal aunt of patients in Cases 1 and 2.

Examination.—This showed a woman who looked much older than the age stated. The pulse was 100; systolic blood pressure, 120; diastolic, 80. There was no von Graefe but a distinct Möbius sign is present. There was general emaciation. The reflexes were lively. Blood examination showed: hemoglobin, 65 per cent.; red blood cells, 3,500,000; white blood cells, 8,000; polynuclears, 60 per cent.; small mononuclears, 25 per cent.; large mononuclears, 12 per cent.; eosinophils, 3 per cent.

Case 4.—*History.*—(Fig. 3). The patient, aged 6, the daughter of the patient described in Case 2, and granddaughter of the patient described in Case 3, was brought to the clinic by request. Her mother stated that nothing abnormal except the enlargement in the neck could be seen in the child. Examination revealed a very large thyroid, and a von Graefe sign, but no other ocular symptoms. Her pulse was 100, and she had mild anemia. The urine was negative. A roentgenogram of this patient was not secured.

Case 5.—*History.*—B. B. (Fig. 4), the daughter of the patient in Case 3 and sister of the patient in Case 2, aged 33, married, has two healthy children, and gives the same family history as the patient in Case 2. The patient states that she suffers a good deal from painful menstruation, but the periods are regular and normal in quantity. At about the age of 25 she began to notice the enlargement of the thyroid gland, and at the same time the hyperthyroid symptoms began to appear. At the time of presentation she showed a distinct von Graefe sign, a distinct Möbius sign, a general enlargement of the thyroid gland, the right lobe being somewhat larger than the left; she had a fine tremor of the hands; a distinct bruit was heard at the neck; the pulse was 130; the heart was enlarged; the systolic blood pressure was 128; diastolic, 70. The urine was negative; the blood count was not made. Roentgenographic examination showed enlargement of the left ventricle. During the examination the patient was in a state of marked perspiration, and showed marked dermographia.

The daughter of Case 3, who underwent operation for enlarged thyroid, was seen by the examiner, but unfortunately complete examination could not be made. It was, however, learned that she had been gradually improving since the operation, though at times she still suffered from cardiac palpitation.

ANALYSIS OF CASES

On analyzing our cases, we see that the mother of the patient in Case 3, the great-grandmother of the patient in Case 1, was a very stout woman. We will not be wrong if we presume that in all probability that woman suffered from a hypothyroid condition. Nature, in order to compensate this condition, gave to the patient in Case 3 the hypersensitive gland which began manifesting undue secretion at middle age. This woman in turn gave birth to children with distinct hyperthyroidal signs which assumed a pathologic aspect in the form of exophthalmic goiter. Here a process of degeneration took place. Nature was apparently making an attempt to eliminate this family. Members of the third generation began to develop symptoms of the disease at the age of 27; in the fourth generation the symptoms began to present themselves in a girl of 6 and in a boy of 10. A natural process of extermination is apparently taking place.

Apparently another factor besides the thyroidal instability is added to the situation of this family, and that is the diabetes in the grandfather of the patient in Case 1. Whether it is due to this factor, or to the instability of the thyroid itself, the fourth generation can no longer procreate, in spite of the fact that the husband of the patient in Case 2, the father of the patients in Cases 1 and 4, is a perfectly healthy man of athletic build and even temperament. Consanguinity in this family could not be detected. A Wassermann test was made in all except Case 4, and every reaction was negative; syphilis, therefore, may be excluded. The transmission was a direct one and along the female line. It may also be emphasized that the occurrence of Basedow's symptoms in a boy, aged 10, and in a girl, aged 6, is in itself an extremely rare condition, well worth reporting.

69 East Ninetieth Street.

THE CURATIVE INFLUENCE OF INFLUENZA IN A CASE OF SPECIFIC MENINGOMYELITIS WITH CYSTITIS *

CHARLES W. BURR, M.D.

PHILADELPHIA

In the older books on medicine, written in what we moderns like to call, perhaps not quite accurately, the prescientific age — before students had become slaves to instruments of precision, when increase of knowledge came only by sharper observation, when chemistry was still alchemy and physiologic chemistry was yet unknown, and when comparative pathology was impossible because the human mind, held in the fetters of religious myth, could not conceive that disease is the same in man and animals — there are many references to the fact that sufferers from certain diseases are immune to certain others. Thus, e. g., victims of gout were well known to be immune to certain other disorders. More or less fantastic hypotheses were called in to explain the phenomenon and, after a time of active interest, there followed a long period in which little or no attention was paid to the matter. Still later came the discovery of the specific vaccines, attempts at explanations of their mode of action, and in recent years, with a widening point of view, an increasing literature on the subject of the curative influence of nonspecific proteins; in other words, the antidotal influence of one infection against another.

Today there is an active interest in this question of the curative, or at least ameliorating, influence of foreign proteins in acute infectious diseases, and renewed attention is being paid to the old subject of the influence of one infection on another. For example, it has long been noted that acute gonorrhea subsides during typhoid fever. Several investigators have noticed that in paresis intercurrent infections are frequently accompanied by a "remission" of the mental symptoms of the disease. The antagonism between carcinoma and tuberculosis has long been known. Von Wagner obtained favorable results from the tuberculin treatment of paresis. He believed that its use increased the favorable action of antisyphilitic treatment, and Donath suggested treatment by abscesses from turpentine injections. Several similar therapeutic measures have been tried, but none has stood the test of time.

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* Read at the meeting of the Philadelphia Neurological Society, Jan. 23, 1920.

The following case is an example of the favorable influence exerted by an acute infection—influenza—on an illness of many months' duration, enabling a bedridden woman suffering from an old meningomyelitis, with severe cystitis, to recover sufficiently to do housework.

REPORT OF CASE

History.—The patient was a housewife aged 36, who came to the Orthopedic Hospital and Infirmary for Nervous Diseases, Aug. 19, 1917. She stated that her illness began in January of that year with constant and severe pain and stiffness at the back of her neck, numbness in both arms, and later pain in both legs. The symptoms slowly grew worse and she became bedridden the following March. Soon after she was confined to bed it became difficult to pass urine and her bowels thereafter moved only by the use of enemas and drastic purgatives. For four weeks she had to be catheterized. At that time also there was complete palsy in her arms and legs, but she had no trouble in speaking or in swallowing. By the end of April she had improved enough to leave bed and was able to stagger around the house, though she still had pain in the right leg and both arms and some palsy in her extremities. After a short time she gradually grew worse. By July she had lost all power in both legs; she was compelled to use enemas, and to have the bladder emptied by the catheter. In the same month she began to have trouble with vision and, for a time, transitory diplopia.

Her past history showed that she had had typhoid fever when 12 years old. Something that was called congestion of the brain followed the birth of her first child. She had five living children and had had no miscarriages.

Examination.—On admission there was absolute palsy of arms and legs with paralysis of the bladder and rectum. Speech was normal. The facies was expressionless and smoothed out, indeed it resembled that of paresis, but during the course of her illness she did not have any of the mental symptoms of that disease. There was tactile anesthesia from a level just above the breasts on the trunk, and on the arms it was present, growing more distinct as one approached the hands. The knee jerks were spastic and ankle clonus and the Babinski reflex were present on both sides. There was no deformity of the spine and no pain on pressure. The heart action was regular and, though the sounds were of poor quality, there were no murmurs. Her blood pressure was: systolic, 100; diastolic, 60. The lungs were normal. Electrical examination of the arms and legs revealed no reaction of degeneration. There were no trophic changes in the skin, but some weeks later a superficial burn caused by a hot water bag was very slow in healing. (Later she had bedsores on the buttocks.) She menstruated several times while at the hospital. By Nov. 10, 1917, she could use the arms quite a little, though still unable to stand. There were involuntary jerking movements of both legs at times, and she had a distinct girdle sense. There was gray atrophy in both eyes and the blood vessels in the fundi were narrowed and thin. The right eye had 5/200 vision, the left could distinguish hand movement. The pupils were irregular in outline and their response to light was very poor. The blood Wassermann test, which was taken immediately on arrival at the hospital, was + + + +. On arrival, and throughout her stay, there was marked cystitis. At times the urine was ropy. On March 16, 1918, the bacteriologic examination of the urine revealed an almost pure culture of colon bacilli and a few staphylococci.

The blood examination revealed: red blood cells, 3,600,000; white blood cells, 15,600; hemoglobin, 0.68. The red blood cells varied much in size and intensity of color. There were 3 per cent. eosinophils. The red corpuscle count and the hemoglobin percentage remained fairly constant, but the number of white blood cells varied greatly from time to time.

Course and Treatment.—The patient had many febrile attacks of varying duration during her stay in the hospital. The temperature would suddenly shoot up to 103 F., then for some days it would be normal and then go up again or swing between 103 and 100 F., and finally after a varying time remain permanently normal for days or weeks. I could find no other cause for the fever than the cystitis and the bedsores. As to medical treatment, she was given potassium iodid up to 300 grains a day; she received large doses throughout the greater part of her stay in the hospital. Mercurial inunctions were used. She had a few injections of salvarsan but without any marked immediate effect. All antisyphilitic treatment was stopped four months before leaving the hospital.

The patient was discharged July 20, 1918. She was then bedridden, completely palsied in bladder and rectum and in the legs and had bedsores on the buttocks. She could use both arms weakly. The urine was thick as cream. I sent her home to die. In December, 1919, her family physician gave me the following account of the course of events: For several months after going home there was no change; then she was seized with an illness, which he diagnosed as influenza. Her fever (102 F.) lasted only a few days and then to his surprise her urine began to clear up and within a few days all signs of cystitis had passed away. At the same time she began to regain control of the bladder. Shortly after power began to return in her legs and after some weeks she was able to stand. Improvement continued for months.

When she came to my office, during the Christmas holidays of 1919, she walked in. Her gait was spastic but she needed no help, not even a cane. She had good control of bladder and rectum. The deep reflexes in the legs were increased. The bedsores were healed. Her arms were normal. There was no anesthesia. The urine was normal. She told me she cooked and did other work in her house and stayed up all day.

COMMENT

The explanation of the mode of action of both specific and nonspecific protein therapy is still hypothetical. Either there is a stimulation of bodily resistance to disease, or a destruction of the microorganisms themselves, and hence a cessation of the production of toxins, or there is a real antidotal effect, i. e., the chemical introduced destroys the poison produced by the micro-organisms of disease.

I suspect that in my patient the long continued cystitis acted as a continuous poison and increased general weakness so greatly as to make it impossible for the patient to use what little of her cerebrospinal motor apparatus remained intact. I also believe the influenza cured the cystitis and thus left the patient with a somewhat injured but workable nervous machine. The original lesion was a syphilitic meningomyelitis, but I am inclined to believe that the antisyphilitic treatment stopped further progress by the spirochetes. I do not, however, propose the artificial induction of influenza as a curative measure for inflammatory diseases of the spinal cord.

Obituary

PHILIP COOMBS KNAPP, A.M., M.D.

Philip Coombs Knapp was born June 3, 1858, at Lynn, Mass., where he received his preparatory education. He graduated from Harvard College with the class of 1878, and entered the Harvard Medical School. He received the degrees of A.M. and M.D. in 1883, after service as house officer at the Boston City Hospital and the Boston Lunatic Hospital, as it was then called.

Among the neurologists of this country who may be said to have influenced medical science should be ranked the strong personality of Dr. Knapp. Besides his scientific attainments he was a scholar and litterateur, with a singularly well-ordered mind, a superretentive and accurate memory, and a large store of learning on a great variety of subjects. He possessed a wide knowledge of current literature, history and friction, and together with a solid grounding in the dead languages was familiar with French, German, Italian, Spanish and even Portuguese. He was an omnivorous reader, a close student of Dante, a writer on scientific subjects in leading magazines, as well as a speaker of excellent diction and a fluent though trenchant debater. As an extensive writer on medical subjects he is perhaps best known. All these accomplishments he enlisted in the service of neurology wherever possible. He was past master in its bibliography.

In 1887, 1895, 1901 and 1911 he acted as editor and cotranslator of the successive editions of Strümpell's "Text-book of Medicine," adding to the last an original section on mental disease. His first work as an author was a volume on "The Pathology, Diagnosis and Treatment of Intracranial Growths" which he published in 1891. He contributed the section on "Nervous Affections Following Railway and Allied Injuries" in Dercum's textbook on nervous diseases, and "Feigned Diseases of the Mind and Nervous System" in Hamilton's system of legal medicine. Few of the aspects of neurology or psychiatry escaped his ready and instructive pen, as the long list of his contributions to medical publications bears witness.

He held numerous positions and memberships, among them: the presidency of the American Neurological Association, the New England Society of Psychiatry and the Boston Society of Psychiatry and Neurology. He was a fellow of the Royal Society of Medicine, and a member of the Neurological Society of the United Kingdom, the New York Psychiatrical Society, American Institute of Criminal Law and

PHILIP COOMBS KNAPP

1858–1920

Criminology, the American Association of Medical Jurisprudence, American Medico-Psychological Association, American Medical Association, and several others. For five years he was a trustee of the Boston Insane Hospital. He was especially devoted to the interests of the American Neurological Association, and was among the foremost of its members in promoting a high standard of membership and quality of work.

Although by nature inclined to be silent and uncommunicative in private, he rarely missed an opportunity to speak—and to the point—at medical meetings which he attended religiously. His opinions, once carefully formed, he held to tenaciously, and was determined and outspoken in supporting them.

As a medical expert he was effective and thoroughly at home. Here his imperturbability stood him in good stead, while his discernment and fund of knowledge of nervous and mental conditions and criminology gave great weight to his testimony. He was consequently in great demand in medicolegal cases, and was highly respected by both bench and bar.

Of Dr. Knapp as a clinician a former student of his, Dr. Cannon, professor of physiology in the Harvard Medical School, gives the following testimony from personal experience:

> Dr. Knapp placed extraordinary insistence on thoroughness. Each one of us in turn had to take charge of the examination of a patient while Dr. Knapp and the rest looked on, and to examine him from top to toe and report with exactness on everything that we found. After that there was a discussion of the interpretation to be placed on the objective findings which was highly illuminating. This experience was one which appealed to me very strongly, and I have repeatedly testified to the excellence of Dr. Knapp's method. Along with it there went in his teaching a dry humor which many of us much appreciated. He frequently made references to novels and other reading, and I recall his testimony that he managed to get through at least a novel a week. I have always regretted that more men did not have the advantage which I had of coming into relations with Dr. Knapp as a student, and deriving from him the discipline which he so capably enforced.

From 1886 to the time of his death Dr. Knapp held the position of physician for diseases of the nervous system at the Boston City Hospital where he soon became noted for his skill in diagnosis. From the first he was indefatigable in his efforts toward the development of that department, and he enjoyed the satisfaction of seeing it grow, through many years and much opposition, from a small outpatient clinic to a separate department for nervous cases on special wards. He was also a warm advocate of the treatment of mental cases on the wards of general hospitals, and was instrumental in having the practice adopted at the City Hospital — an entirely new departure in New England, and with but one or two exceptions in the entire country.

About five years ago he had a hemiplegic attack from which he soon recovered sufficiently to resume work. This he did with his old determination but diminished vigor and impaired motility. Nevertheless he spurred himself on and fulfilled his regular duties, in court, at the hospital, in private practice, etc. Even during the last six months of his life when he became increasingly and plainly incapacitated, he persisted in his efforts with indomitable spirit, only taking to his bed when locomotion became impossible. On Feb. 23, 1920, he died of cerebral thrombosis at the Boston City Hospital where he had served faithfully for thirty-five years as visiting physician. His widow survives him.

Dr. Knapp's strongest characteristic, known only to his intimates, was a constitutional diffidence that bred a certain taciturnity and unresponsiveness, and led him to assume a mental attitude which was at variance with his true nature. Only his closest friends therefore could fully know the warm heart, the loyalty in friendship, the fund of humor and store of knowledge which lay beneath.

HENRY R. STEDMAN, Boston.

Abstracts from Current Literature

ABSTRACTS OF SOME RECENT WORK ON THE NEUROGLIA BY THE SPANISH SCHOOL

The following abstracts are an attempt to summarize in a cursory manner the most important services rendered to histopathology of the central nervous system in the last few years by the Spanish school of histopathologists. Any abstract gives but a pale shadow of the original work and in this case the limitations are much exaggerated by the lack of illustrations which are admirable in profusion, accuracy and beauty in the original articles.

EVOLUTION OF THE NEUROGLIA AND SPECIAL CONSIDERATION OF ITS RELATIONS TO THE VASCULAR APPARATUS.

N. ACHUCARRO, Trab. de lab. de invest. biol. Univ. de Madrid **13**:169, 1915.

The author emphasizes the importance of the theory of the endocrinic activity of the neuroglia, the increased acceptance of the theory (Nageotte, Mawas, Achucarro, Cajal, Collins, etc.) and the prominent position which the glial "foot" attains in the elucidation of the theory, the foot occupying the position of secretory and excretory organ at one and the same time. The development and histologic conditions of the feet, therefore, may serve as an index of the sanguineal-glandular function of the neuroglia.

The author reviews the work on the ontogenesis of the glial foot, naming Held, Rubaschkin, da Fano and especially Cajal who, in a late work, has shown that as soon as vessels appear in the central nervous system glial feet are seen to be formed at the expense of the peripheral prolongations of the epithelial neuroglia, or sometimes at the expense of collateral branches of the peripheral prolongations mentioned.

The author also draws attention to his work on the Ammon's horn where he has stated that the attraction evidently exercised by the vascular system on the glial protoplasm resulting in the formation of feet was manifested not merely on the entrance of the vessels into the nervous structure, but also later, when the vascular system is completely organized (as in a cat of 10 days), glial cells are seen polarized, with their prolongations directed to the surface, that is, to the mesodermic tissue of the pia mater so that their prolongations pass over the vessels without the insertion of feet.

Therefore, the positive attraction of the mesodermal tissue of the pia mater surpasses decidedly at this phase of development the influence which the walls of the vessels exercise on the glial protoplasm, which influence at a later stage becomes predominating. It is then a question as to whether the walls of the vessels or the glial protoplasma are deficient, at this time, in some element necessary for their contact. The author suggests that the vessel walls are not complete at this time.

The author's research is directed to the study of the development of the neuroglia in the different vertebrate groups, with special attention directed to the relations of the vessels and the glia.

He has used two methods only, the Cajal gold method and the tannin-silver method. The *Cyprinus auratus, Labrax lupus, Rana esculenta, Lacerta viridis, Fringella coelebs, Anax* and to a limited extent, the *Amphioxus* were studied by the author.

The author resumes his work in the consideration of these four points:

1. The glia before the appearance of the vascular "feet," the vascular feet of a transitional type and the terminal feet.

In the fish studied by the author, vascular "feet" are not present, although the glia cells are present in numbers around the vessels, and the vascular system is well developed. The ependymal glia traverses the tissues and is implanted on the pia mater. The development of the ependymal glial system is so great and the cells so abundant in protoplasm that one is tempted to admit the existence of a real ependymal gland as such an enormous development could scarcely be considered necessary for the support of the optic roof. The optic lobes in addition possess an external perforative system that is completely independent of the ependymal system. The glial feet implanted on the pia stain perfectly with both gold and silver-tannin methods and are certainly homologous with the glial feet attached to the vessels.

In the Rana esculenta the transitional foot appears. The glial architectonic is the same as in the fish and the relations of the glia to the pia mater are unchanged, but in addition small rounded or fusiform enlargements of glia are seen placed on the vessels in passing over them.

The lizard shows an enormous development of the glial vascular feet although the gliatectonic is still principally epithelial and radiated. But the lizard shows an intermediate stage of glial architectonic as the autonomous development of the feet is in the thalamus opticus and some median nuclei, so that it would seem probable that the evolution of the feet begins in these areas. Intermediate forms are also seen in the lizard, but the majority are terminal.

In the birds studied the relations of glia and vessels are very close and the glia is autonomous. The glial feet are attached to a large part of the vascular surface and form a complete sheath about the vessels. The feet and the sheath present a finely reticulated and vacuolated structure showing that they are not merely for support, but are functional in character. Some of the vascular feet in the bird are elongated and terminate in cones of implantation.

2. Ependymal and autonomous neuroglia.

The author calls attention to the nomenclature in order to draw attention to the ependymal origin of the autonomous glia. The transition from one type to the other is particularly well seen in the lizard. Small autonomous cells, the ependymal glia inserting feet on the vessels and cells detaching themselves from the ependyma and becoming autonomous are seen. In the fish the transitional elements are few, but in the cerebellum examples are seen of the detachment of the ependymal cells.

The evolution of the neuroglia follows and perhaps contributes to the development of the nervous system, a fact that suffices to attribute another rôle to the neuroglia than that of support.

3. Satellite cells and the third element, nonglial (of Cajal, etc.).

The author refers to the recent work of Cajal in which he treats of the real neuroglial satellites and their relations to the nerve cells, and also of the adendritic cells. The author states that a like disposition is not constant in all vertebrates. When the neuroglia is not yet autonomous the satellite cells could not be expected to appear, but even in the lizard when there is an abundant supply of autonomous glia, the satellite cells are not seen. In the *Cyprinus* the author has seen a small number of glial satellites.

The adendritic cells are not seen in the lower fish organisms but are seen first in the white commissures and the optic chiasm of the lizard. It is to be noted that the close relations of the glia to the vessels are first seen abundantly in the same area. If the theory of Cajal and the author is correct, that the adendritic cells originate from the vessel walls or their adventitial sheaths and if, as the author supposes, the tendency of the glia to approach the vessels is due to some change in their walls, it may be that the two facts noted above have a certain relation to each other.

4. Protoplasmatic glia.

Through the studies of Cajal, Golgi, etc., it is known that the ependymal or epithelial glia changes its aspect in passing through nervous structures of different consistency in turn becoming stiff or fibrous, or becoming velvety and protoplasmatic. Müller's cells in the retina furnish the best example, but the same modifications are seen also in the spinal cord and in the encephalon of the amphibia and the reptiles. For instance in the optic roof of the lizard near the ependyma the cells present a vacuolated, protoplasmatic structure; abundant transverse spinous ramifications contribute to the protoplasmatic character of the cell.

The distinction between protoplasmatic and fibrous cells is most important, however, in the autonomic glial system; for the researches of the author and of Cajal show that the persistence in adult age of protoplasmatic cells is a characteristic correlative with the progressive development of the cerebral cortex in the animal series. One must recollect then that the rabbit, the cat, and even the dog, do not present this type of cell well developed. Also in fish the autonomic cells are of a very simple and primitive type; in the optic lobes, at least, they are frankly fibrous.

The lizard shows autonomic protoplasmatic cells, but much like the embryonal cells in the new-born cat. The cells are poor in protoplasma and frequently polarized and no vacuoles are seen. On the other hand, the birds show a structure very like that seen in man. The relations of vessels and glia are correspondingly close in the birds and the impression of a glandular structure is given.

The author considers his work has strengthened the theory that the neuroglia is an endocrinic gland, probably in part, at least, from the beginning of the formation of the neural lobe, pouring its secretion into the tube itself, which at a certain stage of development would open externally by means of a neuropore.

The aspect of the ependymal epithelia in the *Amphioxus* and especially in fish tends to this hypothesis, and the fact that several glands such as the epiphysis and the epithelia of the choroid plexus are developed from the neural tube renders the idea not unnatural. The extraordinary development of the ependyma in certain parts of the encephalon suggests the condensation of this structure into an individualized organ.

Cajal, accepting Achucarro's theory that the epithelial neuroglia of the lower vertebrates is a tubular ependymal gland, attributes to this structure an important rôle in the elaboration of the nutritive milieu of the nervous elements. The ependymal gland and space would thus form the most important source of nutrition for the neurons whose bodies would thus be forced not to abandon the neighborhood of the ependyma and therefore their form would be monopolar. This is the case in the nervous system of the *Amphioxus* and in a different situation it holds true of the monopolar cells of the vertebrates although here the nutritive source is not cerebral, but peripheral at the surface of contact of the ganglions and the mesodermic tissue.

The penetration of the blood vessels into the nervous system of vertebrates of a higher order and the vascular evolution of the glia would allow the neurons to abandon their position in touch with the ependyma or with the periphery, and would permit them to adopt bipolar or stellar forms best adapted to their associative functions.

Two movements in evolution contribute in transforming the glia into a definite vascular gland; first, the insertion of peripheral or mesodermal prolongations of the ependyma on blood vessels; second, the emigration and autonomic transformation of the neuroglia. These two facts provoked, perhaps, by the insufficiency of the nutritive relations of the epithelia with the mesoderm coincident with the increased thickness of the walls of the neural tube at certain points (encephalon) suffice to place the glia, on the one hand, in contact with the vascular system of the nervous centers, and on the other hand, to detach it from its connections with the ependyma.

To admit the secretory functions of the neuroglia, basing the theory on histologic findings, a protoplasmatic structure, young and not fibrous, must be recognized in the cells. Such a structure is plainly seen in the human brain and in that of birds.

The protoplasmatic character alone gives belief in the secretory function and although it is generally admitted that even the fibrous cells are covered with a certain amount of protoplasm, still one must recognize that the secretory function of the neuroglia has developed principally in certain parts of the nervous system and must attain a different grade of importance in different species.

Some light may be thrown on the functions of the neuroglia by the establishment of a histologic fact occurring late in evolution: the close relations of neuroglial satellites to the nerve cells. Thus the neuroglia constitutes an important line of connection between nerve cells and blood vessels. This fact must be of great importance functionally; the neuroglia thus facilitates the nutritive processes of the nerve cells and probably is a factor in the development of the nervous system.

But if with Nageotte, Mawas, Cajal and the author (Achucarro) one attributes an internal secretion to the neuroglia, it must also be admitted that this internal secretion, thanks to the close connection of its producing organ with the nervous elements, must have some function in relation with the specific functions of the nervous system and especially of the gray substance.

Now we know that associative nervous activity produces of itself a certain physical reaction and that this reaction is immensely heightened in processes of affective nature, so-called emotions.

The somatic reaction on the vasomotor muscles, on the glands, particularly, and also on the respiratory muscles and on the muscles of the skeleton is so well known and studied that it is not necessary to insist on its importance. In fact the theories of James and Lange, that the essential and causal moment of the emotions consists in the somatic reactions, has not been entirely abandoned. Whether one accepts this hypothesis, explaining the origin of the emotions or not, one is obliged to admit that perhaps the most essential character of what we call emotions is the physical reaction.

Recent investigations, those of Cannon in particular, have shown the importance of the changes in the composition of the blood in emotions, especially in the action of sugar and epinephrin. It seems to be proved that there is a hyperactivity of the suprarenal glands in emotional states. On the other hand, much more distant studies and the modern studies of Crile admit the stimulation of the activity of the thyroid gland in emotions. Thus two endocrine organs are seen to function in an abnormal manner in the emotions and in relation with respiratory and vasomotor manifestations, which are in turn emotional reactions of such a very sensitive and subtle character that if they are perceived in a subjective manner they are already betrayed by physical symptoms. Apparently these phenomena of expression are as intimately allied to affective psychic processes and in a manner as constant as sensory excitations are to sensations. It would, therefore, seem justified to admit as a working hypothesis that protoplasmatic neuroglia considered as glandular by its structure and its vascular relations, placed in intimate contact on the one hand with the associative nerve elements and on the other hand with the vessels, may act at certain moments of nervous activity and pour hormones into the blood which would serve to set free other endocrinic manifestations essential to the emotions (hyperthyroidism and hyperadrenalism).

NOTES ON THE STRUCTURE AND FUNCTIONS OF THE NEUROGLIA AND IN PARTICULAR OF THE NEUROGLIA OF THE HUMAN CEREBRAL CORTEX. N. Achucarro, Trab. d. lab. de invest. biol. Univ. de Madrid **11**:187, 1914.

The author reviews the specific theories of Weigert, Golgi, Cajal, Lugaro and Nageotte on the functions of the neuroglia. The theories of these men are: Weigert: The neuroglia fills up the interstices between the nerve elements. Golgi: The neuroglia extracts nutritive substances from the blood vessels (by means of glial "feet") and carries them to the nerve cells. Cajal: The neuroglia acts as insulator for the nerve tissues. Lugaro: The neuroglia exercises a double antitoxic function, toward the blood and toward the nerve cells. Nageotte: The neuroglia forms an interstitial gland.

The author formulates the following conclusions derived from his own special methods of fixing and staining the tissues:

1. The neuroglial net (syncytium) described by many (Held, and others) is not shown by the author's tannin-silver method.

2. The protoplasmatic glia cells of the cortex show great numbers of branches which cross at times, but this ramification has not been shown to anastomose.

3. Cajal's method of nitrate of uranium and silver shows pictures similar to Achucarro's. Imperfect stains show a reticulum somewhat like that described by Held, and others, but this reticulum is not in the least like the protoplasmatic ramifications of neuroglia shown in the perfect preparations.

4. Glial protoplasm contains certain granules stained by the silver-tannin method, which stains the mitochondria of other organs brilliantly. Therefore, the granules in the glia may be considered as mitochondria of glia.

5. A certain opposition exists in the number of fibers and the number of mitochondria in glia cells of the cortex. Fibrous transformation of the protoplasmatic cells implies a certain involution of the protoplasmatic activity of the glia, attributing importance to the granules (which have disappeared as fibers are formed).

6. The glial feet stain very perfectly by the nitrate of uranium and silver method. Some feet are seen to send fibers across to the opposite side of the vessel and much beyond it. No continuous membrane is shown to be formed by "feet."

7. The functions of the neuroglia are multiple. The neuroglia assists in breaking down nerve tissue (Alzheimer, Jakob, etc.), in forming scar tissue, and it acts as a support in its fibrous stage. It acts as insulator for the nerve tissues and probably has a secretory function. This secretion may be in relation to the functioning of the nervous system, on the one hand, and in relation to the vessels, on the other, constituting an internal secretion.

CONTRIBUTION TO THE STUDY OF THE NEUROGLIAL ARCHITECTONIC OF THE CEREBRAL CORTEX OF AMMON'S HORN AND OF THE FASCIA DENTATA. N. Achucarro, Trab. d. lab. de invest. biol. Univ. de Madrid **12**:229, 1914.

The author notes the studies already made on the architectonics of nerve cells, fibers and blood vessels, which show that each system is organized in a distinct manner in reference to the architectonics of the other elements. Analogy suggests that the same would hold true of the neuroglia. The one-sidedness of the fiber methods used mainly in the study of the neuroglia up to the present time is mentioned by the author.

His method of tannin-silver is given: Blocks of tissue not exceeding 2 to 3 mm. are fixed from two to three days in a solution of 20 per cent. formol to which sufficient ammonium has been added to make the reaction alkaline. Sections should not be more than 10 mm. thick.

1. Mordant the sections in 10 per cent. aqueous solution of pure tannic acid, in a thermostat at 60 degrees for ten minutes, not allowing bubbles to form.

2. Wash well, after cooling, in aqua distillata with two drops of ammonia.

3. Impregnate in Bielschowsky's silver solution much diluted (3 to 4 drops of solution after precipitate is dissolved, in 10 c.c. water), section by section removing each as soon as the white substance begins to be stained.

4. Reduce in formol solution, prepared as for fixation, for five minutes. After washing the sections may be gilded, then dehydrated and mounted as usual.

The author gives these reasons why the study of the architectonic of the glia is of great value in pathology:

1. The neuroglia reacts very strongly in pathologic conditions and the protoplasmatic cells are frequently transformed into fibrous cells.

2. Certain cortical zones contain only protoplasmatic cells in normal conditions.

3. Certain zones that are protoplasmatic in man are frankly fibrous in the animals customarily used for experiment.

The author's results are:

1. The neuroglial architectonic existing in man and in the monkey, in Ammon's horn and the dentata fascia, corresponds to the stratification of the neuronic structures.

2. In man and monkey in the layers of the stratum lucidum, stratum radiatum and the molecular layer of the fascia dentata the protoplasmatic glial elements predominate, in contrast to the predominence of fibrous elements seen in the dog, cat and adult rabbit.

3. In man, monkey, dog and cat the superficial prolongations of the ependymal cells are not seen in the gold method, but in the rabbit and the new-born cat they are seen in abundance.

4. The irregularities in the course of the ependymal fibers are caused in part by the course of the subependymal vessels, which through mechanical or other unknown reasons carry the fibers and even ependymal cells with them in their course.

5. In some cells in the cat and constantly in the rabbit certain intensely staining granules are seen (in the gold method) which are probably normal components of the glial cells.

6. The perforating fibers in the fascia dentata constitute an embryonal structure that disappears in the adult. They can still be seen in a child of 3 years. In the 10 days old cat these fibers are of great importance, at 3 months they have atrophied considerably, and in the adult cat they have practically disappeared.

7. The cells with perforating fibers lie in the layer of polymorphous cells and in the granular layer of the fascia dentata. Their deep or ependymal prolongations are seen only in some cells of the new-born cat.

8. In the new-born cat scarcely a trace of fibrous transformation of the astrocytes is seen. The polarization of the cells is marked, especially at the level of the stratum radiatum, whence the protoplasmatic pole is always directed toward the surface.

9. The cellular polarization persists in the adult rabbit, and decreases in the dog, cat, monkey and man.

10. Stimulation originating in the vascular apparatus probably collaborates in the definite polarization of the glia cell, and it is probable that the greater polarization of the glia cells in the white matter is determined by the large dimensions of the vascular meshes, which in its turn determines the unilateralness of the attractive stimuli of the neuroglial protoplasm.

11. In the points of contact and adhesion of the two convolutions (fascia dentata and hippocampus) marked variations of the marginal gliatectonic is produced, due probably to the penetration of important vessels and to the development of the Ammon's horn structure. Notable variations in the gliatectonic takes place also at the level of the ependyma through the penetration and division of the vessels.

ALTERATIONS OF THE SUPERIOR CERVICAL SYMPATHETIC GANGLION IN SOME MENTAL DISEASES.

N. Achucarro, Trab. d. lab. de invest. biol. Univ. de Madrid **12**:55 (May) 1914.

The author reviews the histologic work done on the ganglions and also the small amount of research in pathologic conditions, instancing Angiolella's work in general paralysis, Laignel-Lavastine's in Korsakow's disease, Weber's in epilepsy, Spratling and Roswell Park's in senile dementia, G. Sala's in general paralysis with Argyll Robertson syndrome, etc.

The author emphasizes the modern theories of the relation between the sympathetic system and mental processes, and calls attention to the action of the superior cervical ganglions in the innervations of the blood vessels of the central nervous system.

The author's present studies have been made with a variety of stains: toluidin blue, light green, Heidenhain's hematoxylin, etc., but the results to be reported were obtained entirely from Cajal's silver nitrate method, with fixation in ammoniated alcohol.

Two cases of senile dementia, one of arteriosclerotic dementia, two of dementia paralytica, one of juvenile general paralysis, one of Korsakow's, one of epilepsy, and one case of hypothyroidism in a young patient were studied.

Certain difficulties in the research were encountered as no equivalent picture was found equalling the Nissl structure in the cortex.

Notwithstanding these difficulties, two elements at least vary sufficiently in the various diseases to be worthy of study. These elements are the pericellular tangles and the structures of the protoplasm and morphology and structure of the dendritic prolongations.

Cajal considers these pericellular fibrillary tangles as abnormal although not of necessity caused by any diseased condition. They are in his view either results of degeneration or of attempts at regeneration. The author's researches bear out this view. He notes hypertrophy of the fibers of the tangles, formation of rings of fibrils, variation of the tangles in number and also in the complexity and in the number of turns of their spirals. A second histologic alteration is a degeneration of the protoplasm of the cell with vacuolization. The process extends to the protoplasmatic dendrites.

After citing these alterations in the various diseases studied, the author concludes that the tangles or spirals are abnormal and that they vary in number and in size in pathologic states. The vacuolization also varies in extent sometimes passing on to complete destruction of the cell. Vacuolization to an extreme degree was noted in Korsakow's disease. The pericellular fibrillary tangles were profuse in general paralysis and exceptionally so in epileptic degeneration.

THE CORTEX IN CEREBRAL PARALYSIS. N. Achucarro and M. Gayarre, Trab. d. lab. de invest. biol. Univ. de Madrid **12**:1 (May) 1914.

1. The gold method of Cajal is especially adequate for histopathologic investigations on account of the extent and constancy of its staining properties.

2. The cortical astrocytes undergo alterations in their structure, in general paralysis, resulting in the disappearance of their spongioplasm and the diminution of the gliosomes.

3. The hypertrophy of the cortical astrocytes implies a simplification of the protoplasmatic ramification. While some branches are much thickened, others become pale, atrophic and fragmentary.

4. Neuroglial cells terminating in expansions, rounded or club-like, are frequent in general paralysis.

5. (Klasmatodendrosis of Cajal.) Fragmentation of the glial dendrites is common in general paralysis and results in some cases in the formation of ameboid cells and also of "full Körperchen" of Alzheimer.

6. The process of fragmentation does not intervene in the formation of the small ameboid cells close to the nerve cells. The latter cells stain with gold and differ from the adendritic cells of Cajal which do not take the gold.

7. The connection of the glial satellites with the nerve cells becomes closer in general paralysis, the glial cells forming more or less continuous sheaths. The satellites are distinguished in three groups, those of the base, of the neck and of the trunk of the nerve cells, together forming the sheaths.

8. Although the adaptation of the glial cells to the nerve cells is very close, the nuclei of the glia cells remain round and never become so large as those of the rod cells.

9. The rod cells in general paralysis as in rabies do not stain by the gold method. These results and those of the silver-tannin method, which is so elective for connective tissue, tend to show that the greater part of the red cells are mesodermal in origin, coming from the vessels, and, according to the authors' opinions, migratory in habit. They cite the irregular curved forms which Alzheimer calls transitional as evidence of a drawing away from the point of origin and assume that the cell only becomes a complete rod cell when freed entirely from its origin.

CONTRIBUTION TO THE STUDY OF THE NEUROGLIA IN THE HUMAN CORTEX IN SENILE DEMENTIA AND OF THE PARTICIPATION OF THE NEUROGLIA IN THE CELLULAR ALTERATION DESCRIBED BY ALZHEIMER (ALZHEIMER'S TANGLES).

N. Achucarro and M. Gayarre, Trab. d. lab. de invest. biol. Univ. de Madrid **12**:67, 1914.

The authors take as their point of departure Alzheimer's classical description: "In the interior of a cell, which appears otherwise normal, one or several fibers appear unusually clearly on account of their special thickness or their density of impregnation. At a later state many adjacent fibrils alter in the same manner and thus form thick bundles that finally appear on the surface of the cell. Finally, the cell with the nucleus is entirely destroyed and the point where the cell lay is indicated by a bundle of fibers rolled up into a spiral." Alzheimer also noted that chemical changes, shown by staining reactions, accompanied the morphologic alterations. The authors then cite the work of observers who are not in agreement with Alzheimer's theory: Bonfiglio, Donaggio, Marinesco, Cajal, Tello and Achucarro. They discuss particularly Achucarro's work of 1910, in which he showed that fibrillary "baskets" were formed by glial elements and at times were rolled around the nerve cells; he concluded at that time that some (not necessarily all) of the "tangles" in Alzheimer's disease had their origin in the neuroglia. Bielschowsky advanced the same idea later, drawing his conclusions from the similarity of certain vascular connective tissue changes to the Alzheimer lesions. Simchowicz is cited as the most open opponent of the ideas of Alzheimer. Simchowicz was not acquainted with the work of the Spanish school, but came to the conclusions, from his investigations, that the tangles were in all probability neuroglial in origin, but at all events that they were found outside the nerve cell. The authors do not consider Simchowicz' theory, that all tangles are neuroglial, proved since to prove the nuroglial origin of the tangles, it would be necessary to show continuity of the tangles with the prolongations of some glial cell. This phenomenon has not yet been demonstrated, Achucarro's work showing only that structures resembling the Alzheimer tangles can originate from changes in the neuroglia. The difference in chemical constituency of the glial cell and the tangles has been offered as an explanation of the apparent lack of continuity, but the fact that Bielschowsky's method stains both elements and yet no connection is seen between them, speaks against this supposition. The author's results with Cajal's gold method form an argument against the neuroglial origin of the Alzheimer baskets and tangles.

The gold method stains the neuroglia intensely, but the neuronic elements are also stained a deep rose or violet color and the skeins or tangles of Alzheimer are palely perceptible. In pictures shown by this method, however, the neuroglia appears to act in the ordinary manner, passing over and around the nerve cells, sometimes passing through the meshes of the "tangles," but never taking any part in their formation. From time to time, however, structures very similar to the Alzheimer lesions are seen, but on close observation a glial nucleus can be found in the midst of the fibrillary tangle. These elements stain intensely with all silver and gold stains, whereas the tangles of neuro-fibrillary origin stain palely with the gold method.

After citing many cases and conditions observed the authors conclude that: 1. In senile dementia the glial elements undergo alterations that may give origin to figures similar to Alzheimer tangles. 2. Cajal's gold method gives no indication that the neuroglia takes any considerable part in the formation of the typical Alzheimer changes, and still less that the tangles are exclusively of neuroglial origin. 3. The Alzheimer figures resemble, more than anything else, the phenomena observed by Cajal after section of the central axon, when globular masses are formed at the peripheral extremity very similar to the spirals or tangles. Cajal considers them agonal formations. 4. Alzheimer's phenomenon is essentially a retrograde and agonal manifestation of the nerve cell, in whose terminal stage a special incrustation exists that may attack neuroglial structures. 5. In the final phases of Alzheimer's phenomenon portions of the cell undergo a process of excision and remain separated from the rest of the cell.

Gurd, Ann Arbor Mich.

(*To be continued.*)

AUTONOMIE DE LA MOELLE CONSECUTIVE A LA SECTION COMPLETE DE L'AXE SPINAL. G. Marinesco, Rev. neurol. **35**:257 (April) 1919.

For a long time before the war, the foremost theory in regard to the autonomy of the spinal cord separated from the brain, was that of the distinguished English neurologist, Bastian, who stated that complete section of the spinal cord at the level of the upper dorsal and lower cervical region produced total paralysis of the lower limbs and abdominal muscles, disappearance of feeling below the lesion and loss of the cremasteric and abdominal reflexes and also loss of the tendinous reflexes, the only reflex still persisting being a slight movement of the toes after deep pricking. This theory had many supporters in France, Germany and England.

The neurology of war, however, has brought to hand new material which makes us believe that the spinal cord severed from its connections with the brain has an independent activity. On close examination we find that the cases published by Bastian and his followers to support his theory are open to discussion. To be valid, the following conditions should be true: first, the patient should live long enough to allow the spinal cord to regain its own activity, that is, from four weeks to several months; second, the elements of the reflex activity should be intact—radicular cells, motor nerves and muscles. Electric examination can inform us of the state of nerve and muscle. Third, we should have accurate details on the condition of the radicular cells in connection with the patellar and Achilles' reflexes; fourth, of the condition of the motor nerves of the crural quadriceps and of the triceps surae; and, fifth, we should know the histologic condition of the quadriceps and especially of the vastus internus muscle.

In none of the observations published by Bastian or his followers are all of these conditions fulfilled. Many times they are cases of compression of the spinal cord rather than of true spinal transsection. In the opinion of the author these observations show definitely only two things: that superficial and deep reflexes disappear immediately after the complete section of the spinal cord, and that it is possible for this disappearance to persist ultimately because some part of the reflex arc has been injured.

Opposed to the theory of Bastian, we now have a number of facts which show that the isolated spinal cord does regain a reflex activity after a certain length of time; namely, material presented by Kausch, George Riddoch, Lhermitte and Roussy and a case of M. Poussepe's in Petrograd which six months after the operation for suture of the meninges showed important modifications: pinching the dorsal surface of the foot was followed by a movement of triple contraction of the legs on both sides. In this movement of contraction, the tonus of the shortening muscles was exaggerated. To this movement of triple contraction of the excited member there corresponded a movement of crossed extension produced in an almost constant manner.

It is unquestionably true that the sectioned spinal cord after a certain time regains its own activity which is not a continuation of the old but a new and lower activity, lacking the activity posture and reproducing in only a rudimentary way the activity of the lower animals. It is manifested in the reappearance of the tendinous and of some defensive reflexes and the return of their tonus. The extensor muscles, which depend on the supramedullary centers, do not regain their reflex activity. It all falls within the province of the flexors.

The first question, that is, the question of the nature of this activity, the writer discusses by an examination of its fundamental characteristic, tonus, so unequally distributed in the flexor and extensor muscles. It might be called the expression of a stimulation which gives birth to a slight tetanus. It is physiochemical rather than physiologic in nature, for chemical phenomena such as nutritive exchanges do take place, though they are very small, thus accounting for any absence of fatigue. To better understand the delicate mechanism of tonus, the author here discusses the researches of Botozzi on physiologic tetanus which led him to the belief in the dualism of the substratum of the muscle substances—sarcoplasm, the seat of tonic, and the muscle fiber, the seat of clonic contraction.

Many experiments have been made on the condition of the current of action in muscular contraction. We have Pipper's theory of voluntary innervation; that each muscular fiber involved in tetanus is traversed each second by fifty waves of contraction. There is an accordance between the impulsions in the nerve and in the muscle. He also shows that the normal rhythm of innervation is a coefficient of the temperature. The oscillations increase with the temperature, as is shown in an interesting experiment on the muscles of cold-blooded and warm-blooded animals. These have the same rhythm if the temperature is equal. There is, then, a general law for the current of action or waves of negativity in the animal series.

When the tonus is modified, modifications in the central nervous system would extend to the electric phenomena of the nervous influx. These oscillations, we find, diminish in number in the case of hypertonia as in that of hypotonia, but this again is always followed by a lowering of temperature.

DuBois-Raymond has always stood as authority in experiments in electrophysiology and was the first to try to determine the mathematical formula in his attempt to relate the muscular contraction to the intensity of the stimulation. But, after all, what is important, is not the strength of the intensity of the current, but the speed of excitability of the muscle, the rapidity with which the intensity of the constant current is attained and suppressed: "chronaxia," as M. Lapicque calls it, which is a constant of time.

He defines it more accurately a little further on as the inverse of the speed of excitability. The muscle and its corresponding motor nerve have the same chronaxias, are isochronal. When a nerve and muscle are isochronal, any disagreement in their chronaxias makes difficult their harmonious action. When the chronaxias are unequal, the nerve stimulation is transmitted with more and more difficulty to the muscle; when the harmony of the chronaxias is equal to one half, the stimulation of the nerve is no longer transmitted. In partial hemiplegia the chronaxia of the muscles is slightly altered. It is increased in muscles contracted in hemiplegia; the temperature is also lowered. Chronaxia is a function of temperature. In sections of the spinal cord, chronaxia is altered, since the temperature of the muscles of the lower limbs is decreased and the tonus changed.

The writer believes that in the activity of the spinal cord freed from its connections with the brain, we have to deal, not merely with the phenomena of shock, though the suppression of certain cerebral organs may account for the degeneration of the radicular cells, but with modifications of a physical order as well, such as differences in superficial pressure of the cells and in electric potential. These physical modifications: superficial pressure, electric potential and heat, bear strongly on chronaxia and destroy the harmony that exists between the synapses of the higher centers and the medullary nerves. Therefore he thinks that the spinal cord separated from its connections with the brain, shows after a certain period an activity of its own. It is the seat of elementary reflex phenomena, but this lower activity is abnormal and does not correspond to the activity of the lower animals; the agreement between the neurons of the flexor muscles and the sensitive neurons has returned, which accounts for the movement of triple contraction mentioned in the case of M. Poussepe, a trace of a coordinating mechanism of the swimming movements of the duck. But the agreement between the extensors, which depend for innervation on the higher centers, and their corresponding neurons, no longer exists. They do not regain their reflex activity. Their tonus does not return to normal.

The fact that we are dealing with a new activity of the spinal cord does not mean that regeneration of the sectioned fibers does not go on. It has been proved that these fibers do possess the capacity for regeneration. But this does not assure the return of any useful connections with the brain, nor does it explain all the activity of the sectioned spinal cord. It is the activity of the flexors and their neurons, the return of their tonus, and the lowering of temperature of all the muscles, that make the writer believe that we have to deal with a new and autonomous activity of the spinal cord, shown by defensive movements, so ill-adapted, however, that they cannot be classed with the true reflexes of defense of the lower animals.

Jones, Detroit.

TRAUMATIC NEUROSIS AND TRAUMATIC HYSTERIA. Michael Osnato, Neurol. Bull. **2**:334 (Sept.) 1919.

Osnato bases his study of traumatic neurosis on a series of 102 cases which were for a long time under his care in connection with the State Industrial Commission of New York. The vast majority of his patients were married men, most of whom received some injury to the head; more than half were unconscious following the accident, and nearly all showed marked emotion of fear at the time of the injury and more than 80 per cent. immediately developed "nervous" symptoms. The most common symptom complained of was sleeplessness, frequently alternating with terrifying dreams; diffuse headache was another common complaint; and dizziness (not true vertigo) completed the usual picture. Physically the examinations were invariably negative, except for lively reflexes; and endocrinologically the author was unable to detect any incriminating evidence. Mentally the patients uniformly showed mild depression or anxiety, many of them betraying facial expressions of fear and exhaustion.

Osnato insists that most of his patients were previously normal; they betrayed no true mental abnormality during the periods of their incapacity. Most of them had no apparent reason for prolonging their illness, certainly not for pecuniary reasons, as the compensations they received were invariably less than they could have earned by going back to work. The author is firmly of the opinion that persons suffering from traumatic neuroses have sustained real injuries, and he seeks a causal explanation in anatomicophysiologic and psychologic substrata.

He refers to Mott's investigations of the functional nervous conditions of the war, to Pike and Coombs' study of traumatic shock, to Crile's description of structural changes in the brains of animals and persons subjected to prolonged fear, and to Cannon's researches on shock; but Osnato is not convinced that anatomicophysiologic explanations are sufficient to account for the traumatic neuroses that he encountered in his civilian practice. He therefore turns to psychologic considerations and finds in MacDougall's conception of the instincts and their emotions a truer explanation of the genesis of traumatic neuroses. The crux, as Osnato sees it, is this: The injury is accompanied by intense emotion (fear); but soon this emotion ceases to be associated directly with the accident and becomes attached, perhaps by a circuitous route, with other psychic processes which may have had some relation with the original excitant of the fear reaction but which no longer stand in direct or indirect relation, and cannot even be recalled by the patient to have had such a relation. In terms of physiology one may find an analogy in Pavlow's conditional reflex.

As most of the cases resulted in litigation they were followed up after settlement and of twenty-five cases investigated, twenty-one had apparently recovered. Osnato thinks that trauma can produce functional nervous disease in persons who had previously had no mental ills, and he deems it wise to make early and suitable settlements lest the neurosis become fixed and adjustment impossible.

Referring to the traumatic hysterias, Osnato calls attention to the fact that while the immediate causal factors were similar in them and the neuroses the patients were of different mental stuff and therefore reacted differently. He studied seventy-three patients; most of them had previous mental abnormalities, frequently difficulties in the sex or social life. One point of difference was that in traumatic hysteria the symptoms gradually grew worse up to the time of settlement, then slowly receded. The symptoms, and particularly the common signs of hysteria, were not amenable to treatment before settlement. Psychologically, the hysterics differed in that many were emotionally unstable, asocial, sensitive, selfish and below normal mentally. Their complaints were not genuine, they were insincere and tried to misrepresent. Frequently it was difficult to differentiate them from true malingerers particularly so as the causative mechanism lay close to the surface. In nearly all cases the desire to obtain a settlement motivated the syndrome.

By way of illustration the histories of eight cases are detailed in which previously existing social or sexual maladjustments, and not the accident, conditioned the hysterical manifestation.

Osnato concludes that in the traumatic neuroses one deals with normal persons whose symptoms are brought on by the accident and fear, and who actually suffer a psychophysical depression, while in the traumatic hysterias the accident is merely an "excuse" for bringing to a head previously maladjusted instincts. In the opinion of the author, one never sees true hysterical stigmas in the traumatic neuroses.

In the study of his cases Osnato has attempted to distinguish two distinct clinical manifestations, each silhouetted against distinct personality backgrounds.

Wechsler, New York.

AN ACUTE EXHAUSTION DREAM STATE WITH PSYCHANALYTIC INVESTIGATION.

Prof. Giovanni Marro, Lieutenant-Colonel, Consulting Neuropsychiatrist, Military Hospital Grenoble, Arch. di antrop. crim. **39:** Pt. 3-4, 161, 1918-1919.

The patient was an interned Austrian capitalist of great power and wealth captured by the Italian troops with the surrender of Monfalcone in 1915. He was interned in Alexandria and allowed considerable liberty. A former sweetheart from Turin came to see him, and for three "days and nights they abandoned themselves to great sexual excesses." After the departure of the woman the patient became nauseated and vomited and had fever with much sweating. He drank a good deal of water but took hardly any food for several days. He refused medical attention. Eight days after he had first seen his sweetheart he awoke early in the morning, took a train to Turin, visited her there, rented a small apartment and then went to the police notifying them that he had chosen Turin as a residence with the consent of the authorities in Alexandria. Asked why he desired this change he stated that he "had killed two persons in Alexandria who had broken into his lodgings to revenge themselves on him because he had denounced them as thieves. They tried to kill both him and his host. The authorities had absolved him from blame because of legitimate self defense but advised him to leave Alexandria and go to Turin to live." The authorities in Turin, realizing his abnormal mental state sent him to the insane asylum where he appeared stunned and indignant. He was weak and feverish and resisted but little being put to bed. When seen by the doctor he repeated his story, insisted that peace had been declared between Austria and Italy because his father and mother had visited him in the hospital the night before and told him so. He was imperious in his demands to be set free. His fever left him in a few days, his appetite returned and he was much quieter. Finally one day he exclaimed, "Why, I have dreamed all this."

Before recounting the dreams the author goes into a detailed description of the patient's physical characteristics according to the classifications of physiognomy of Lombroso. After the acute delirial dream state the patient was quite depressed, diffident and apprehensive. He recovered in a month and was discharged.

In the analysis the author recounts six dreams that the patient had. These the author thinks motivated the patient's conduct and controlled his waking state. He believes with De Sanctis that the paranoic dreamer believes absolutely in the reality of his dream states, and he thinks with Macario that the delirious dream states are so infixed that in medieval days they caused such staunch belief in their reality that sufferers resisted tortures. Discussing the latent content of his patient's six dreams, the author states that the subject was able himself to see points of contact between the meaning of the dreams and his affective life as he was living it, a captive in an enemy country. In his dreams the patient gave free play to his emotions and desires—peace for his fatherland, relief from financial difficulties caused by his position as a prisoner, relief from the agony of seeing and hearing a victorious Italy, etc. His inadaptability, sense of isolation and antagonism resulted in a lack of contact with reality and a flight into his acute exhaustion delirium. The author calls attention to the fact that between each of the six dreams there was a waking period of from a few minutes to a whole day, but the content of the dreams was emotionally so attuned to the patient's personality that it controlled him by subjecting him to the dominance of each separate dream. Each dream completed and dominated the subsequent mental state. In the fifth dream he emerged triumphant and victorious in direct physical combat killing his enemies and surmounting all difficulties. As the patient says "if I was late in persuading myself that the whole thing was a dream, it was because I did not wish to think it was—in fact, I always tried to chase the thought that it was a dream, out of my mind." The freudian explanation is that the dreams were able to control consciousness by eluding the censor because they so nearly identified themselves with the ego.

Osnato, New York.

ATROPHIE OPTIQUE FAMILLIALE. S. Imamura and K. Ichikawa, Rev. neurol. **35**:277 (April) 1919.

The authors present two cases of optic atrophy with mental symptoms occurring in brother and sister. Five other siblings did not exhibit a similar condition. The family history was negative and neither parent had suffered a disorder similar to that exhibited in the two cases reported.

The authors give the eye findings and clinical history of the two patients. In both the papillae were rather limited in circumference and pale throughout their extent. There existed an absolute central scotoma of from 30 to 40 degrees in the case of the brother, more extended in the right eye than in the left; and scotoma of from 10 to 15 degrees in both eyes in the case of the sister. There was no contraction of the peripheral visual field.

In the course of hospital treatment for the girl, the papillae showed signs of inflammatory atrophy. Amblyopia slightly increased. In both eyes, a relative scotoma of 10 degrees appeared around the absolute scotoma which remained unchanged. The peripheral limits of the visual field became contracted. The slight change in the absolute scotoma and in amblyopia lead to the conclusion that the disease had reached its terminal stage.

The authors attempt to classify these cases according to the known types of optic atrophy. The opthalmologic picture in both cases reproduces almost exactly the symptoms of hereditary optic atrophy as presented by Leber. But these patients exhibited other neuropsychopathic signs not included in the type of Leber.

In the case of the brother, the lack of coordination of the ocular movements and asymmetry in the innervation of the face, were not sufficiently marked to be important. The first might be a result of the weakened vision; the second, an hereditary defect. Intellectually, he was, to all appearances, normal.

But in the case of the sister, one cannot ignore the following neuropsychopathic symptoms: lack of responsibility, smiling nonchalance, mental slowness, weakened memory, feeble innervation of the right side of the face and appearance of fibrillary tremors on the lips and eyelids during mimic movements. On looking at a distance, one eye turned downward. The convergence was imperfect. Tremors in different parts of the body appeared, exaggerated in the upper members during purposeful movements. There was rigidity in all the members, more marked in the upper body. All movements needing fine coordination were ill adjusted and slow. No ataxic phenomena were present. Romberg's sign was absent. The gait was almost normal. There was no alteration in the deep and superficial reflexes. Mental deterioration began at the time of the appearance of the disease.

In regard to the hereditary optic atrophy of Leber, Hormuth has shown that while dizziness, headache, scintillating scotoma and other symptoms are included, anything of a hemopathic nature, such as epilepsy, is considered an exception. It is doubtful that the authors' case belongs to the type of Leber. Raymond distinguishes Leber's disease from the other optic atrophies, by the name of hereditary optic atrophy of the papilla; the others, he calls, hereditary atrophy of the optic nerve.

There is one type of optic atrophy, however, that includes nervous phenomena and with which we may connect our case. It is a type presented by Behr and Kiel, concerning a series of six children, all boys, who exhibited signs of pyramidal and cerebellar lesions in a slight degree: hypertonia of the limbs, exaggeration of the reflexes, slightly pronounced ataxia, uncertain gait, a touch of nystagmus and weakness of the bladder. The authors call this "complicated infantile familial optic atrophy," as opposed to Leber's type which they call "uncomplicated juvenile familial optic atrophy." Six other cases from the same clinic are added, which present the paracentral scotoma, in all of which the nonprogressive character of the disease is established. All these cases, however, concern only children, and are mostly hereditary, while in the authors' case the symptoms date from the period of puberty. With the accumulation of more material, it is probable that the transition between these two types of Leber and Behr will some day be established.

Jones, Detroit.

CHRONIC NONDEGENERATIVE HEREDITARY CHOREA. I. S. Wechsler, Neurol. Bull. **2**:329 (Sept.) 1919.

Wechsler describes a case that seems to him to represent a distinct clinical entity although it bears close resemblance to Huntington's chorea.

The patient was an American woman, aged 36, who had been suffering from peculiar movements of the arms, hands, legs and body and twitchings of the face for sixteen years. The movements were intensified by emotion and were said to be present even during sleep. She also complained of peculiar spells of weakness.

Her family history was interesting. Her father had had a chronic chorea for twenty years before his death at 68, which was due to tuberculosis. Mentally he was sound. One brother had "shakings." She had four children: one daughter of 18 had nystagmus; another of 16 had nervous spells; one boy of 9½ had chorea which had lasted for three years; another boy of 6 also had mild chorea which began at the age of 3. None of the patients showed mental deterioration at any time though the chorea had persisted for a number of years.

The woman showed the following signs of chorea: She had a swaying, jerky, irregular gait, the whole body being incoordinate and clownish. Coordination was poor, speech slow and jerky, but not scanning. There were irregular, jerky, purposeless, meaningless, quick movements of whole parts, of arms, legs, body and hands. She could not hold her head in one position, the eyeballs wandering in an irregular, jerky, choreic way. The face showed an occasional grimace and the tongue was quickly jerked back on momentary protrusion. Further neurologic and systemic examinations were negative. The mental status was normal.

The whole picture, Wechsler claims, closely resembles Huntington's chorea though there are a number of points which sharply distinguish the two. There is a hereditary element, three generations showing prolonged chorea, but while his patient undoubtedly had a chronic chorea it was not progressive or degenerative, and therefore he thinks it does not belong to the Huntington type. He ruled out hysteria despite her peculiar spells of weakness, which bore a marked functional stamp, and the intensification of her movements on emotion. Chorea gravidarum and paramyoclonus multiplex are considered only to be dismissed. Chronic perennial chorea, chorea adultorum permanens and chronic intermittent chorea are mentioned merely as descriptive names which Wechsler does not think fit his case.

In view of the fact that the patient's condition was hereditary, chronic, not progressive and not mentally degenerative the author thinks it cannot be considered a Huntington, even though the clinical picture closely resembles it. Whether or not it is a distinct clinical entity, he claims, is an opinion which does not as yet admit of proof.

Osnato, New York.

CHEMICAL ANALYSES OF TWO PATHOLOGICAL HUMAN BRAINS. C. G. MacArthur and E. A. Doisy, Am. J. Insan. **76**:159 (Oct.) 1919.

The authors report the results of analyses of two brains, one from a case of paresis and the other from a case of organic brain disease, probably bulbar paralysis. The Wassermann reaction with the serum from both cases was positive.

For purposes of analysis the brains were divided into cerebrum, cerebellum and brain stem (including medulla, pons, midbrain and thalami). The cerebrum from the case of paresis and the entire brain from the case of bulbar paralysis were analyzed. The results are tabulated together with those from three normal brains. The ages of the pathologic brains were 50 and 51 years and those of the normal brains 33, 35 and 67 years. The tables are arranged to show the constituents in percentages of fresh tissue, in percentages of solids and in weights or constituents in grams. Total solids, phosphatids, cerebrosids, sulphatids, cholesterol, organic and inorganic extractives, lipin, protein, neutral and inorganic sulphur and lipin, protein, organic and inorganic phosphorous were determined. The authors summarize their findings as follows: The principal changes in the cerebrum of the case of paresis were in the lipins and extractives. The substances (cerebrosids) associated most closely with sheath development showed largest losses. The phosphatids and sulphatids are present in smaller amounts than in normal cerebrums. Both organic and inorganic extractives showed marked increases in percentage and absolute amounts. This is to be expected in degenerative changes, if it is the more complex structure substances that alter to simpler compounds which are but partly removed from the tissue.

The changes in the cerebrum in the case of organic brain disease (probably bulbar paralysis) which gave a syphilitic history showed essentially the same kind of changes as the case above; but all were less marked. This would suggest that the alterations were due to the action of spirochetes. The midbrain, medulla and pons exhibited about the same kind of chemical changes as the cerebrum. The cerebrosids, sulphatids and phosphatids, and therefore the total lipins, showed distinct losses. The proteins, water and total solids were nearly normal; but both organic and inorganic extractives were high. Though the data for the cerebellum show variations, they are not supposed to be of pathologic significance.

Weston, Warren, Pa.

INFLUENCE OF PITUITARY EXTRACTS ON THE GENITAL TRACT.

Robert T. Frank, M.D., J. A. M. A. **73**:1764 (Dec. 6) 1919.

The author reports briefly the results obtained from the feeding of pituitary extracts to white rats. Assuming that anterior lobe extracts cause an increase in the growth and stimulation of the genital tract, a series of experiments was conducted in order to determine what constituents of the pituitary extracts produced these phenomena. The portions of the gland experimented on were: Extract No. 1, anterior and posterior lobe in proportion of 1 to 6 desiccated; Extract No. 2, pituitary free from fat soluble substances; Extract No. 3, pituitary containing only fat soluble substances; Extract No. 4, anterior lobe desiccated and diluted with lactose to prevent decomposition.

Six litters of white rats raised in the laboratory were used in the course of these experiments. The rats were 3 weeks old when the experiments were begun. Litters 1 and 2 were each composed of six sisters; Litters 3 and 4 of five sisters each; Litter 5 of three sisters, and Litter 6 of four sisters. At least one animal was selected as control for its group. For thirty-five consecutive days each animal (except the control) was fed 0.05 gm. of pituitary extract, given in a bolus composed of meat, egg and cream cheese, which was eagerly taken. In order to round out the diet, the animals were also fed with milk, greens and oats. Throughout the experiments all the animals, except No. 4 of Litter 6, showed progressive increase in growth.

At necropsy, no concordant increase in size, general body weight or in the genital organs could be found which was ascribable to the glandular feeding. Irrespective of whether or not they were experimental animals or controls, certain rats showed an increase in weight, the presence of corpora lutea in the ovaries and enlargement of the other internal genitals.

In conclusion, the author states that in his opinion further experiments will be necessary to prove that the stimulation of the female sex organs can be ascribed to the use of pituitary extracts. He also emphasizes the danger of using glandular extracts in clinical practice, since laboratory reports have not been confirmed.

Adson, Rochester, Minn.

THE CEREBROSPINAL FLUID IN MULTIPLE SCLEROSIS.

Joseph Earle Moore, M.D., Arch. Int. Med. **25**:58 (Jan. 15) 1920.

The positive colloidal gold reaction is found in a large number of patients with neurologic disorders of all types. The "paretic" gold curve, supposed to be pathognomonic of general paralysis, has also been obtained in other conditions—lead poisoning, tuberculosis, meningitis, brain tumors and multiple sclerosis; in multiple sclerosis it appears quite regularly, but in the first three conditions it is less regular.

The records of forty cases previously reported present no uniformity; fourteen gave a paretic gold curve, twenty-five were negative, one case was atypical, fourteen showed pleocytosis and in twenty-five cases the globulin was positive. The Wassermann reaction for blood and spinal fluid was negative in all but one case which showed a doubtful reaction.

The author reports twenty cases of multiple sclerosis in which the diagnosis was definite. Eighteen of these showed a definite paretic gold curve and positive globulin reaction. The average is high enough to exclude an occasional finding. In eight cases in which the diagnosis was doubtful no reactions were found, showing that the diagnosis was probably at fault. Therefore, excluding the occasional finding of the paretic gold curve in lead poisoning, tuberculosis, meningitis and brain tumors, there are two conditions — neurosyphilis and multiple sclerosis—in which it is found with constancy. In differentiating these two we have the positive Wassermann reaction in either blood or spinal fluid, with one single exception in long treated cases in which the Wassermann reaction becomes negative before the colloidal gold test.

Therefore, when the clinical evidence points to multiple sclerosis, the spinal fluid picture with a paretic gold curve and positive globulin would be a strong argument in favor of a definite diagnosis of multiple sclerosis.

DeLong, Philadelphia.

TRANSPLANTATIONS TENDINEUSE DANS LA PARALYAIE RADIALE TRAUMATIQUE. Presentation d'un Cas. T. Tarkowsta and H. P. Achard, Rev. neurol. **35**:283 (April) 1919.

The great interest which the war has excited in operations on the nerves, often brilliant and successful, has led us to neglect for the time being, the surgery of the tendons. The authors believe that transplantation of the tendons would be of benefit in many cases where operations on the nerves have originally failed. To draw attention to this field of surgery, they present the following case:

A soldier was wounded July 18, 1919, by a bullet in the left arm, with shattering of flesh, fracture of the lower third of the humerus and immediate radial paralysis. After removing the patient to a hospital, the wound was cleaned and dressed, roentgen-ray pictures taken and the arm immobilized in a plaster cast.

When the cast was removed, about September 14, a complete neurologic examination was made and a diagnosis of radial paralysis with involvement of the supinator longus was reached. Total reaction of degeneration was noted, and hypesthesia in the region of the radial nerve on the dorsal surface of the hand; no tingling could be provoked near the fracture nor below it for the length of the radial nerve. No regeneration of the nerve resulted after months of electric treatment. Under the conditions present in the arm, an operation on the nerves seemed a dubious procedure and the authors resorted to transplantation of the tendons. The operation is described in detail.

Ten days later the plaster cast was opened. The hand was in hyperextension and all effort at bending the hand and fingers caused great suffering and was made difficult by the shortened extensor tendons. However, after several weeks of postoperative treatment, consisting of massage, electric treatment, etc., the patient could grasp even very small objects. The movement of extension appeared quickly, produced first by the elasticity of the common extensor muscle, and later by the action of the transplanted muscles.

The patient continues to progress and can bend and extend the hand with ease, although the movement is still somewhat limited; he can use it for all the ordinary needs of life, bend and extend each finger separately, and touch the other fingers with the thumb.

The technic of different operative methods is discussed and that adopted by the authors explained. They used the three longitudinal, cutaneous incisions, chose the great palmaris and anterior cubital muscles for transplantation and passed from the palmaris to the dorsal surface around the radius and ulna.

They conclude from their observations, that in the case of peripheral paralysis, particularly radial paralysis, recourse may be had to transplantation of the tendons when recuperation of the nerve function is doubtful, and that these transplantations are already sufficiently regulated for successful current usage.

Jones, Detroit.

THE PIGMENTATION OF NERVE CELLS. II. THE LIPOCHROME A PLANT PIGMENT. David H. Dolley and Frances V. Guthrie, J. M. Res. **40**:295 (Sept.) 1919.

From the facts that species whose tissue fat is colored with carotinoids carry the pigments in the bloodstream and that species whose fat is colorless do not carry carotinoid pigments in their serum, except in insignificant traces, the authors constructed the working hypothesis that the fat combined or fat-holding pigment of the nerve cells would be a true plant lipochrome or carotinoid, absorbed with vegetable foods and incorporated in the cell but limited to such species as carry carotinoid in the blood. The blood serum of man contains carotinoid pigments.

Taking up the work of Palmer and his associates on the production of pigment-free egg yolk by the elimination of carotinoid containing food from the diet of the chickens, the authors determined the effects of diet and depression on the lipochrome content of nerve cells. Twelve chickens were used. Six were made carotinoid-free and six carotinoid-full, by adjusting the diets. In three of each set the factor of depression was introduced by the use of morphin, exposure to temperatures of 40 to 45 degrees or by inadequate diet. The chickens which had received a carotinoid-containing diet, including both the normal and the depressed group, showed the presence of yellow pigment. The tissues from the morphin-depressed chickens contained an excessive amount of pigment. No pigment could be demonstrated in the nerve cells of chickens fed a carotinoid-free diet.

It was then necessary to show that the pigment found in the carotinoid-fed chickens was identical with the lipofuscin, "Abnutzung" or fat-holding pigment so frequently described as existing in nerve cells. The authors did this by using the ferric chlorid, hydrogen peroxid, Nile blue and solubility tests. They therefore conclude that the lipochrome pigment of the nerve cells is a plant carotinoid derived from the food and incorporated in the cells but limited to such species as carry carotinods in the blood.

Weston, Warren, Pa.

A STUDY OF THE COLLOIDAL GOLD REACTION AND ITS CLINICAL INTERPRETATION. Margaret Warwick and Charles E. Nixon, Arch. Int. Med. **25**:119 (Feb.) 1920.

The authors present a full review of the methods of preparing the colloidal solution and lay especial emphasis on securing exact neutrality. In the study of the cases presented, the blood and spinal fluid Wassermann reaction, the globulin content of the spinal fluid and the spinal fluid cell count were all carried out in conjunction with the colloidal gold reaction. A cell count, if over 5, was considered pathologic since in the entire series of "miscellaneous" or normal spinal fluids the average cell count was 1.6.

The abridged experiences of twenty-five writers are cited *a priori.* These indicate that the colloidal gold reaction is the most sensitive of all spinal fluid tests, but that it tends to show curves in nonspecific fluids and more rarely fails to show specificity where it exists.

The tests were carried out on all hospital cases suggesting central nervous system involvement of any kind, and on a series of "miscellaneous" hospital cases for control.

In dementia paralytica the colloidal gold test was positive in 94 per cent. and the spinal fluid Wassermann reaction positive in 83 per cent. (eighteen cases).

In tabes dorsalis the colloidal gold test was positive in 80 per cent. and the spinal fluid Wassermann test was positive in 52 per cent. (seventy-four cases).

In cerebrospinal syphilis, the colloidal gold test was positive in 85 per cent., the Wassermann test in only 48 per cent., and globulin (Nonne) in 63 per cent. (twenty-seven cases).

In multiple sclerosis, the colloidal gold test was positive in 55 per cent., the Wassermann test in 0 per cent., the Nonne test in 28 per cent. (twenty cases). Here most of the cases fell in Zone 2.

In tumors of the brain and spinal cord, the colloidal gold test was positive in 55 per cent., the Wassermann test in 0 per cent., the Nonne and cell count were both low (fourteen cases). The curves occurred generally in Zone 3. The authors regard the findings as significant and probably diagnostic.

Miscellaneous cases showed twenty-one curves of 240 fluids examined. Twelve of the cases were in central nervous system involvement of some kind. Eight were in syphilitic patients, three of whom showed central nervous system involvement. From these figures the authors conclude that the absence of a curve definitely rules out central nervous system involvement of any nature.

In a small series of treated cases the colloidal gold reaction was found to have no value either in diagnosis or prognosis. The curve may change or remain unaltered and does not parallel the clinical course. The curve may appear with or without other signs after provocative treatment.

Haines, Philadelphia.

THE SPINAL FLUID IN PRIMARY AND SECONDARY SYPHILIS.

Joseph McIver, M.D., J. A. M. A. **73**:1765 (Dec. 6) 1919.

The author, in his study of ninety-one cases of primary and secondary syphilis at the Philadelphia General Hospital, endeavored to determine, so far as possible, what percentage of cases would show infection of the cerebrospinal fluid by the usual laboratory methods. The tests made on each specimen of spinal fluid were the Wassermann, protein determination and cell count, and in every case comparison of the blood Wassermann was made with the findings in the spinal fluid.

The lesions varied from early chancre to a fading secondary rash. A + + + + Wassermann reaction of the blood serum was obtained in every instance; this was one of the factors determining whether or not studies would be made of the spinal fluid.

Only two of the cases showed a slight excess of protein in the cerebrospinal fluid; one of these, curiously enough, was in the chancre stage; the other was of two years' duration. Very little treatment had been given in either case.

The cell count was made immediately on removal of the spinal fluid. In cases of primary syphilis, the average number of cells to the cubic millimeter was 9—the greatest number of cells to the cubic millimeter for any one specimen was 17. In the cases of secondary syphilis, the cell count varied from 10 to 90. If a cell count of 5 to the cubic millimeter is considered the average in normal spinal fluids, practically all these cases showed a slight abnormality.

In conclusion, the author states: 1. There is a slight increase of lymphocytes in the cerebrospinal fluid in the majority of cases of primary and secondary syphilis. 2. The increase in protein is not so frequent as the increase in lymphocytes. 3. In the present series a + + + + Wassermann reaction was not obtained on the spinal fluid in any case of primary and secondary syphilis. 4. It does not seem reasonable to conclude that we can determine by the examinations of the cerebrospinal fluid in cases of florid syphilis, in what cases symptoms of the central nervous system will develop.

Adson, Rochester, Minn.

A NERVE SYNDROME OF THE RETRO-PAROTID REGION. M. F. Bonnét-Roy, Bull. méd. Par. **33**:807, (Dec. 27) 1919.

The comparative rarity of the condition warrants attention to the writer's clinical report.

The case given is that of a woman who developed an acute left-sided tonsillar abscess; the purulent infection extended and led to a focus in the left parotid region requiring external incision and evacuation. Signs of the following nerve injuries were found shortly subsequent: paralysis of the left hypoglossal nerve, paralysis of the left recurrent laryngeal nerve, partial paresis of the left spinal accessory with weakness of the sterno-cleido-mastoid and trapezius, and lastly, left-sided paralysis of the soft palate. Thus, only the last three cranial nerves were affected.

The writer considers that the paralysis was peripheral in type in each instance, and that it was the result of local toxic injury to the nerves.

He offers the subsequent course in the case as additional evidence. There was steady improvement, and two months after the first examination only remnants of the above syndrome remained. The patient was not syphilitic.

Davis, New York.

THE SURGICAL TREATMENT OF GUMMATOUS OSTEITIS OF THE SKULL. A. W. Adson, M.D., J. A. M. A. **74**:385 (Feb. 7) 1920.

The author states that gummatous osteitis occurs in late syphilis and involves the outer table of the skull alone, or the outer and inner tables plus the dura and the brain. The gumma may be an isolated one, but it is more likely to be associated with numerous lesions on the skull, these lesions varying in size from 0.5 to 4 cm.; the smaller ones present a fluctuating mass, while the larger ones are necrotic, suppurating ulcers of the bone and usually open spontaneously.

The author feels that in addition to specific treatment, local surgical treatment, consisting of the removal of the sequestrum, or dead bone, is necessary. He reports a case of brain abscess, on which he operated. Wound infection developed, resulting in a retraction of the skin edges and exposure of the parietal bone over an area of about 9 cm. by 6 cm. Considerable difficulty was encountered in effecting granulation, when the child accidentally bumped its head, loosening the outer table of the exposed bone, which was lifted off. Granulation quickly followed.

The same principle has since been applied in four cases of gummatous osteitis, that is, elevating and freshening the skin margins and removing all the necrotic bone with a chisel or rongeur to a depth at which living bone was exposed. After the removal of the sequestrum, wet dressings saturated either in boric acid or saline should be applied, and if epidermization is slow, skin grafting may be resorted to.

In making a brief summary, the author advises the removal of all necrotic bone in gummatous ulcers of the skull and suggests that this treatment also be applied to areas of denuded bone in nonsyphilitic cases, since granulation will at all times be hastened by the removal of the outer table of the skull.

AUTHOR'S ABSTRACT.

DISSOCIATED AMYOTROPHIC PARALYSIS OF THE BRACHIAL PLEXUS (UPPER TYPE) FOLLOWING THE ADMINISTRATION OF ANTITETANIC SERUM. JEAN LHERMITTE, Gaz. d. hôp. **92:** No. 67 (Nov. 4) 1919.

Lhermitte, after recalling the several manifestations of "Serum Sickness," reports three cases of paralysis and atrophy of some of the shoulder and arm muscles (cervical 5, 6 and 7). The deltoid, serratus magnus, spinati, biceps, brachialis anticus and triceps were attacked. There was no change in sensation in any of the cases, with the exception of severe neuralgia.

The onset was four, five and eight days after the injection. However, Lhermitte does not believe the paralysis was anaphylactic as no previous injections had ever been given these patients. He emphasizes the frequency of the occurrence of neurologic disease resulting from the administration of antitetanic serum.

Lhermitte feels that he has described a new syndrome characterized by a peculiar type of muscular and musculosensory dissociation.

KRAUS, New York.

SHOCK. ROBERT DUPONT, Progrès méd. **34:**519 (Dec. 27) 1919.

The author says it is possible to classify several types of shock. The nervous type is a reflex paralysis of the bulbar centers, the second type follows hemorrhage, the third type—so-called toxic shock—includes both infectious toxic conditions and muscle trauma toxic conditions unrelated to infection. He considers it proved that injured noninfected muscle tissue loses its nitrogen to the blood and "the augmentation of nitrogen substance in the blood (nonprotein and residual) is in direct ratio to the toxic (shock) phenomena."

But is he keeping to a proper conception of shock? Is he not discussing toxemia? The difference should be emphasized. Even as regards his own grouping, he admits that the types cannot always be clinically separated. However, he insists that the muscle injury type (without infection) can occur alone.

His summary concerning treatment brings out nothing new.

DAVIS, New York.

ACUTE ASCENDING (LANDRY'S) PARALYSIS AFTER AN ANTITYPHOID VACCINATION. G. Guillain and J.-A. Barré, Ann. de méd. **6**:218 (Aug.) 1919.

These authors report the case of a soldier who, one day after vaccination against typhoid, fell ill with stiffness of the legs. This spread to the upper extremities. Five days later the palsy became flaccid and the next day involved the bulbar centers. Four days later, the patient died. Kernig's sign was negative. The spinal fluid was negative as to cells (15), increase of globulin, or the pressure of the anaerobes or aerobes.

In brief, this was a case of Landry's paralysis of ten days' duration ending in death. Gross changes in the nervous system were not found at necropsy. No microscopic examination was made.

Kraus, New York.

COMPARATIVE STUDY OF THE BORDET-WASSERMANN REACTION ON THE BLOOD AND URINE. M. Clement Simon, Bull. de l'Acad. de méd., Par. **33**:73 (Jan. 20) 1920.

The author having discovered, in accord with other investigators, that the urine of syphilitics sometimes contains substances capable of deviating complement, has carried out the Bordet-Wassermann reaction on both the blood and urine of a series of 201 patients.

Although in the primary stage, the positive reaction is obtained in the urine in only 16 per cent of the cases, it increases in occurrence in direct ratio to the duration of the infection. In the primary and secondary stages, discordance in the two tests has always been in favor of the blood (blood +, urine —). But in the tertiary stage, the discordance has sometimes been in favor of the urine (blood —, urine +). Likewise in latent syphilis, the discordance was in favor of the blood in twenty-four instances and in favor of the urine in ten. These findings seem to indicate that the urine and blood show serologic differences that closely resemble those existing between the blood and spinal fluid.

Because in nine cases out of 100 a positive test of the urine was obtained when the blood was negative, the author thinks it should be a therapeutic dictum that the treatment of any case should be pursued until there is a negative reaction of the urine. He considers this just as important as bringing about, if possible, negative spinal fluid and blood reactions.

Davis, New York.

THE LEG SIGN—NEW OBJECTIVE EVIDENCE OF PARALYSES OR PARESES DUE TO PYRAMIDAL TRACT DISTURBANCES. J.-A. Barré, Presse méd. **27**:793 (Dec. 24) 1919.

The Barré sign is elicited on the prone subject by flexing the legs to a right angle with the thighs, and then instructing him to retain them in that position. When a pyramidal tract lesion is present, the affected leg quickly drops, either steadily or jerkily. Rapidity and completeness of descent is considered indicative of the severity of the lesion; an incomplete reaction, where the extension stops at an obtuse angle, or is followed by flexion back to the right angle, is still weakly positive. When questionable, the subject is told to flex the legs as strongly as possible on the thighs; flexion should be better on the sound side, and a decisive fall should then occur on the suspected side when the legs are again placed in the vertical position. Another characteristic of a positive reaction is the presence of conspicuous muscular contractions on the affected side, voluntarily opposing the tendency to fall.

Normal persons can hold the vertical position for a long time, and paralyses from other than pyramidal tract lesions are claimed to give a satisfactorily negative reaction, even in the presence of considerable atrophy. Evidence is presented to show that malingering is difficult, and that hysteria gives negative reactions. Five hundred neurologic cases in all were tested, and positive reactions found only in those with at least a slight pyramidal tract disturbance. Pure peripheral lesions gave only negative reactions.

Barré's experience leads him to attach particular importance to this new sign. He believes it to be more valuable than the Babinski or other signs now in common use—more delicate, and as significant when negative as when positive. The explanation of its mechanism offered is that the muscles flexing leg on thigh are the weakest of the lower extremity groups, and that these weak muscles are relaxed while the strong extensors are afforded their optimum position. Further investigation and statistics of this sign would appear well worth while.

Huddleson, New York.

JUVENILE PARESIS. E. L. Hunt, Am. J. Syph. 4:104 (Jan.) 1920.

The author recognizes six types of paresis: the simple dementing, the simple depressed, the agitated, the taboparetic, the classic expansive type and juvenile paresis.

The first reported case of juvenile paresis was that of Clouston in 1877.

Characteristically there is normal development during the first few years of life, with a gradual onset of symptoms that vary widely and may simulate (mental) backwardness, imbecility or cerebral atrophy. There may be defective memory, excessive childishness, fabrication or dementia. Associated with the fabrication there may be the characteristic imaginative embellishments. Sometimes there are alternating periods of exhilaration and depression with fears and anxieties.

The physical symptomatology may comprise tremors, exaggerated reflexes, fixed and unequal pupils and epileptiform convulsions. As in the adult, the multiplicity of lesions affords a widely differing syndrome. Frequently there is rapid emaciation and exhaustion.

The onset is usually at the age of 10 or 12, showing that there is a definite period of incubation in the child as well as in the adult. The course is about four years and the prognosis necessarily fatal. The colloidal gold reaction is sometimes the only means of making the diagnosis.

The patient whose case is reported, developed headache at the age of 6 and one month later had convulsions affecting the left side with severe twitchings of the left arm and leg. The attacks occurred at monthly intervals. A year after the onset there was a convulsion affecting the right side and producing aphasia; before this the patient was irrational and garrulous. The pupils reacted sluggishly to light and accommodation, the knee jerks were increased, there was a slight papular eruption on the forearms and the back. The Wassermann reaction was positive.

The first injection of salvarsanized serum caused an improvement. One other injection was given without modifying the picture. The child died two months after the first injection. No necropsy was performed.

Haines, Philadelphia.

HISTOPATHOLOGIC FINDINGS IN DEMENTIA PRAECOX. Eva Rawlings, Am. J. Insan. **76:** (Jan.) 1920.

The result of a painstaking microscopic study of the cortex in twelve selected cases of dementia praecox is given in text and plates. A uniform set of changes is described: disappearance of stainable substance in the cell bodies, atrophy with fragmentation of the neurofibrils in all body and prolongations, laying down of fat granules and gliosis, most marked in the frontal and central regions. Such studies should be repeated on control cases, though the work involved seems unending, and in all conclusions must be considered the possible changes produced by the more immediate causes of death.

Bond, Philadelphia.

CONVULSIVE CRISES OF PLEUROPNEUMONIC ORIGIN. A. Barbé and R. Glénard, Progrès méd. **35:**113 (Jan. 11) 1920.

Five cases, all in adults, are briefly cited in which convulsive seizures began following either pleuritis or pleuropneumonia. The authors consider that this was more than a coincidence, and believe that the convulsive features were occasioned in each instance by the chest pathology acting as a peripheral irritation. They compare the mechanism to that of the convulsive crises in infants due to helminthiasis. At most, they claim that a pleuropneumonic affection plays an occasional rôle in the etiology of convulsions.

Davis, New York.

Society Transactions

THE PHILADELPHIA PSYCHIATRIC SOCIETY

Regular Meeting, Nov. 14, 1919

CHARLES S. POTTS, M.D., *President*

A CASE OF TRAUMATIC HYSTERIA. Presented by DR. M. R. GABRIO.

J. H., a white boy, aged 12, was admitted to the Philadelphia General Hospital, June 20, 1918. He was transferred to the Camp for Imbeciles at Byberry City Farms shortly after his admission to the hospital, with the diagnosis of imbecility, complicated with convulsions.

The family medical and social history was not obtainable, as the patient had no close relatives or friends and was a charge of the city. The history during his stay in the hospital was: He possessed the intelligence of a rather high type imbecile; clean and tidy about his person; adaptable to his surroundings; and had epileptic convulsions at infrequent intervals, from which he suffered no inconveniences other than at the time of the attacks.

On July 20, 1919, during a severe electric storm, the patient was found lying on the bathroom floor in a semiconscious condition. He claimed to have been struck by lightning while getting a drink of water, and to have been knocked to the floor. He stated that the lightning struck him on the left side of his face, his left arm, forehead and hand. Dr. Gabrio saw the patient within fifteen minutes from the time of the accident. He was in a highly nervous state and complained of pain along his arm and hand. His arm and forearm exhibited a spastic condition which was more marked in the forearm; the fingers and thumb were drawn tightly into the palm of the hand, while the distal phalanx of the thumb was in the position of extension. There was slight impairment of sensation. There were no other physical signs. The pupils were slightly dilated but reacted to light and accommodation. There were no evidences of gross cerebral lesions or any evidence of external injuries, such as burns or bruises. Examination was rather difficult as the patient complained of pain as soon as the parts were touched. Rest in bed with sedatives and massage was ordered.

On July 21 he was again seen. The pain had subsided and the spasticity and marked flexion of the fingers could be partially overcome when his attention was diverted. At this time the psychic and nervous symptoms were not so pronounced, as he was more calm and cooperative. There was still slight impairment of sensation along the arm, forearm and hand. Massage and mental diversion were prescribed, and the patient made a complete recovery in about ten days.

The case was regarded as one of traumatic hysteria resulting from psychic fear or actual shock of the flash of lightning. There is considerable doubt as to the patient having been actually struck by the lightning. The condition was similar to those psychic and physical manifestations seen in shell-shock and certain morbid neuroses.

A PSYCHOSIS IN A MAN WITH SYPHILITIC INFECTION: PROBLEMS. Presented by DR. DANIEL H. FULLER.

The patient was a man, aged 32, a native of Philadelphia, and born of American parents. His father, paternal grandfather and one paternal uncle were heavy drinkers. His father died of cirrhosis of the liver at 44, and one maternal uncle died of pulmonary tuberculosis at 21.

The mother was an energetic, active and capable woman, emotionally unstable but devoted to her children and especially to this only son, whose career she had apparently attempted to dominate in her ambitions to have him succeed. She was argumentative, persistent and determined, inclined to misquote statements and to put words into the mouths of her advisors to color her own views. Her methods might justify, to some degree, her son's complaint that she nagged him and interfered with his affairs, although she was sincere and earnest in her procedures, for he was her pride and his failures were a great blow to her ambitions and a stimulus to her determined effort to help him in her own way and in spite of his opposition. These characteristics of the mother are enlarged on to show a little of the environment to which the patient had to react.

The patient was the second of four children. He started school at the age of 5, entered the high school at 13, the University of Pennsylvania at 17, and was graduated as an engineer at 21, in 1908. The Reading Railroad Company was then elevating their tracks and he was put on that work. He held that position for over a year, then became restless, felt there was no future in the city's employ and too much graft; so he left and took a position with the Pennsylvania Railroad. At the end of three months he was offered a position with the government as inspector of material for the locks in the Panama Canal, at $125 a month; he accepted it and went to Panama in February, 1910, remaining there until September, 1913. He did well and was earning $225 a month when he came back and invested in bonds the $3,000 which he had saved.

On his return from Panama he joined his mother, who had broken up her home and was traveling, and he stayed wherever she happened to be. He complained of being very tired and decided to rest for a time before taking another position. His mother thought this was doubtless the effect of the climate in Panama, and felt it was wise for him to rest; months went on, however, and patient showed no inclination to work; though he was normally restless in disposition, ambitious and anxious to be busy, he was now content to sit around hotels. He was not extravagant in his tastes, did not drink at all but smoked to excess. After his money had been spent, his mother allowed him $12 a week and bought his clothes for him. This seemed to satisfy him; he never complained and never tried to get a position, but seemed a little depressed.

In February, 1916, the patient returned to Philadelphia and took a room in a lodging house on Arch Street, where he met a Danish girl, a divorcée, quite uneducated, who worked in the kitchen of a large department store. He married her on short acquaintance. He had no position at the time but shortly after obtained one with the Pennsylvania Railroad at $80 a month. Although his mother was not pleased with the marriage, she was delighted that he had gone to work, tried to put her disappointment aside and to think that it might be an excellent thing for him. To encourage them, she furnished a house for them. Previous to this, however, and soon after the patient's marriage, the mother offered to help him secure a divorce if he desired at any time to obtain one. The wife gained knowledge of this offer and was naturally much incensed.

In August, 1917, he took a position as government inspector with the Ordinance Department in West Virginia, at $200 a month, where he remained until May, 1918, when he became restless and dissatisfied and miffed over an argument with his superior officer concerning pay for overtime. He was transferred to the Navy Yard in Philadelphia, where he remained three months, until August, 1918. In that position he was reported to have been dilatory and unsatisfactory in his work, and was discharged for inefficiency. He almost immediately secured a position with the Emergency Fleet Corporation at a smaller salary, and remained there until November, 1918, when his present breakdown occurred. His work there was below the grade a man with his education and training should have been doing; he was slow and worked long hours; he was very quiet and had little to say, "was thought to be a 'queer duck' and they let him alone"—no one felt that he knew him at all well.

He had frequently received substantial aid from his mother since his marriage; sometimes it was entirely unsolicited, although very welcome, and many times it was in answer to his appeals.

He is said to have been unusually bright in school and his mother described him as gentle, affectionate, thoughtful and considerate of her, companionable and confiding. A friend of his student days described him as "very quiet, studious and clean-spoken." He had never used alcoholic liquor but had smoked to great excess. He admitted he was told that he had had syphilis some fifteen years ago.

His married life, he said, was happy and without unusual friction until the present trouble, when he claimed his mother interfered and broke up his home. His wife stated that he was always at home, that they had had no quarrels before the present illness, and that he was always reading and studying. They had one child, nearly 2 years old.

The general breakdown dated from November, 1918, when he experienced some kind of a collapse when alone in the office. He described it to his mother as "losing hold," a "sort of fear," and said that he prayed to God to take care of him. His wife came and took him home. She reported that he was found crying with his head on his desk; he thought some "psychologists" were trying to get him to do or say certain things and were working on his mind. He seemed to have recovered in a few days, but did not return to work. His mother was summoned and immediately took him to a nerve specialist, who made a thorough study of the case, obtained a positive Wassermann reaction of his blood and gave a guarded prognosis, advising arsphenamin treatments. The patient consulted several physicians and was finally persuaded to go to a private sanatorium for treatment, his mother agreeing to pay his expenses and also to support the family while he was being treated. He stayed only a week and left in impatience and resentment because his mother had not paid anything to his wife and because he had not received a treatment. Much pressure was brought to bear on him to persuade him to receive treatments, and he finally submitted to four injections of arsphenamin and he absolutely refused more. He was obstinate in his refusal and became bitter, suspicious, resentful and threatening toward his mother and the physicians who urgently advised intensive antisyphilitic treatment. He was sullen, unreasonable, argumentative, and quarreled with his wife, accused his mother of trying to separate them, accused the mother and doctors of a conspiracy to poison him with treatments and to compel him by fair means or foul to take them. He refused to work, demanded $1,500 of his mother as due him for failure to keep her promise, and talked in a most bitter and vindictive manner.

In the hope of compelling him to receive treatments, his mother finally withdrew her financial aid until his wife was forced to store and sell her household goods and go to work to support herself and the child. To learn more definitely of his home life and conduct, the mother's lawyer employed neighbors to observe and report on their goings and comings. This was known to the patient later on. He was finally so suspicious and so convinced of a plot against him, that he personally threatened with violence the consultant physician and also threatened to bring suit against him and his mother and others. At this point his mother was persuaded to have him committed, with the expectation that when committed treatments would be forced and cure would follow. He was committed to the Pennsylvania Hospital May, 1919. Here physical examination showed a body of masculine type, the face symmetrical, the forehead high, the distance between the eyes wide, the features clear-cut and of rather delicate mold. The palatal arch was rather high, the tonsils not unusual, and the teeth in fair condition but needing attention. The heart was negative except for a faint short murmur at the apex, replacing the second sound. The genitals were normal, except for the absence of the frenum which he said was burned off with acid seven years ago. The pupils were unequal, the right being permanently larger than the left; they were regular in outline and concentric with the iris, and reacted promptly to light and accommodation. All of the deep and superficial reflexes were very active. There were no pathologic reflexes and no ankle clonus. Coordination tests were well performed and test phrases were satisfactorily done. There was marked tremor of the closed eyelids, and fine fibrillary tremor of the protruded tongue. The urine showed a faint trace of albumen; the blood Wassermann test was positive; the spinal fluid Wassermann test was negative; the cell count was 1 per c.mm. Protein examination showed the Nonne test to be slightly plus, the Ross-Jones doubtful, and the Noguchi negative. The colloidal gold test read 1111000000.

Mentally, the picture did not change since his admission to the hospital. Consciousness was clear. He showed no special defect of memory and retained his school knowledge. He had a fair knowledge of current events and was clearly oriented for time, place and persons. Emotionally, he was neither depressed nor elated. Affectivity appeared to be diminished, and while he said that he was worried, his feeling appeared to be resentment over his alleged wrongs rather than worry, anxiety or grief over his present situation or his future. He expressed no concern over the support and comfort of his wife and family, and there was marked indifference over matters that would naturally perturb one; nevertheless, he appeared pleased when his little 2-year-old child visited him and was attentive to her at these times. Any questions as to explanation of his past conduct in not working brought out resentful objection to the questions or sarcastic flings at others in his attempts to justify his course. Several times, entirely spontaneously, he told events in the family life which threw suspicion and inuendo on his mother's character and aired unnecessarily certain aspects of the family life in an unfeeling or malicious manner. He at first associated quite freely with others, staying about with them and playing games, but later secluded himself, staying in his room except at meal times, occasionally going to a theater or moving picture show.

There were no hallucinations. His delusions were a mixture of suspicious inferences and willful misinterpretations, which crystallized into the belief that doctors were against him, that doctors ruled the community, that they had deliberately poisoned him and that they could be hired to do any mean job under the cloak of their profession. All his thoughts seemed to be tinctured with a sense of injury and injustice and an irrational determination not to yield in his opinions or to admit reason in those of others. He was quick tempered and on the slightest provocation reacted out of all proportion to the cause in a tirade of abusive talk, vituperative and sarcastic utterances, unwarranted accusations and extravagant statements. He not only lacked insight into his condition and situation, but was unable to discuss it with sense or any approach to fairness. His talk was coherrent and his associative processes clearly established, but his reasoning was weak and foolish and inconsistent. He was reasonably careful of his personal appearance, cleanly in his personal habits and not inclined to obscenity or vulgarity in conversation.

To sum up, a man of 32, with a college education and a record for unusual brightness in his studies, of a quiet, studious and gentle nature, was graduated at the University of Pennsylvania as an engineer at 21. After filling two or three minor positions, he went to Panama and worked hard and successfully as inspector of material for locks on the Canal for over three years. On his return he was disinclined to work after a reasonable rest, and for nearly two and a half years he remained idle, used up his savings and depended on his mother for support. He was lacking in interest and ambition or sense of obligation. At the end of this period, after much pressure to get him to work on the part of his mother who was ambitious for and perhaps oversolicitous and overzealous in her methods, he married a woman of inferior education and social status, although of good character. The marriage was an unconcealed disappointment to his mother. He immediately found employment and continued to work for about two and a half years, but with decreasing efficiency, changing his situation three times and finally breaking down entirely at the age of 32, when it was found that he had a positive Wassermann reaction of the blood. After that he did no work, even though his unproductiveness necessitated the breaking up of his home and the driving of his wife to supporting herself and her baby. He submitted to four intravenous treatments under great protest, but otherwise obstinately refused treatment of any kind.

He was committed to the Pennsylvania Hospital in May, 1919, and while he showed marked physical improvement and acknowledged feeling rested, he was unreasonable, deluded about his condition and the situation, suspicious, abusive and threatening in his talk and obstinately unyielding in his attitude and opinion. There were no hallucinations, no marked memory defects, no euphoria or depression, but a marked emotional apathy in regard to his family obligations. Physical signs were generally lacking except for inequality of the pupils and some fine fibrillary tremor of the protruded tongue. Attempts at diagnosis left him in the unclassified list. Paresis and dementia praecox seemed the most probable considerations.

In view of the gradual development with personality changes and failing efficiency covering a period of six years, culminating in a complete breakdown while at work of lower grade than his qualifications fitted him for, his inability or unwillingness to get employment afterward, his suspiciousness, delusions of persecution, unreasonableness, indifference about his family obligations, great irritability, lack of insight, exaggeration of his own opinions and intolerance of others, one was inclined to diagnose his case as one of dementia praecox of a paranoid type in a man having a syphilitic infection. If he were a paretic, a more pronounced deterioration of both his intellectual and affective faculties would have been expected in connection with the marked changes in personality which so gradually developed and which showed no improvement in spite of greatly improved physical condition. There was not the gross deterioration that usually develops in a much shorter time after the appearance of the first marked symptoms of mental change in the paretic, nor had the physical signs of degeneration appeared.

Problems.—This patient was sent to the hospital by his mother for antisyphilitic treatment with the idea firmly fixed in her mind that arsphenamin injections would positively cure him, and that without these he was doomed to rapid deterioration and hopeless insanity. The patient refused to take treatments; he violently protested whenever they were mentioned, declaring that the hospital had no legal right to force drugs into him, and that he would resist and smash all of the apparatus in sight. He believed that his father was poisoned by mercury prescribed by doctors, and said that he himself was made very ill by the four treatments received the previous winter; he refused also on the ground that he would not take treatments under compulsion of agents paid by his mother, but thought he might consent to take them outside, some time in the future, say a year hence, if he could employ his own doctor and pay him himself. He not only refused intravenous injections but medicine of any kind. He even refused to have his teeth examined and treated, saying he would wait until he was released and then employ his own dentist.

The hospital took the position that it was unwilling to force treatments on a patient who so strenuously and persistently protested while his consciousness was unclouded. There was an uncertainty as to whether the psychosis was dependent on his syphilitic infection, and as no guarantee of cure could be given even if an intensive antisyphilitic treatment was administered, physicians in attendance felt still less inclined to attempt to force him to take such treatment. His mother was equally persistent and determined that he should have treatment, and was critical of the position taken by the hospital. She felt that it was his right and that to protect his best interests and assure him of a future, the treatments should be given in spite of his protests, even if it were necessary to etherize him to do it. Her accusations of lack of cooperation, of failure to give the patient proper treatment, coupled with her earnest beseechings, made the problem more complicated. From the legal standpoint, the hospital was undoubtedly taking a proper stand. With threats for habeas corpus and suits for damages in the patient's mind, there were further reasons for conservatism in the management of the situation, although we believed that intravenous treatment was indicated and would have given it if we had not been opposed.

Was the psychosis dependent on syphilis? Whether it was or not, was it the duty of the physician to compel intensive antisyphilitic treatment against the positive and violent protest of the patient?

CHICAGO NEUROLOGICAL SOCIETY

Rgular Meeting, Nov. 20, 1919

Lewis J. Pollock, M.D., *President*

TUMORS OF SPINAL COLUMN CAUSING NEUROLOGIC SYMPTOMS. Presented by Dr. D. B. Phemister.

Almost every type of new growth occurring in bone may develop in the spine, but their relative frequency differs greatly from that in the long bones. Of the benign tumors the most common is cartilaginous exostosis, which may occur alone but usually is part of multiple cartilaginous exostoses. The tumor may arise from the posterior part of the body or arch of the vertebra and project outward, making root symptoms as in Figure 1, or it may project into the spinal canal where it makes compression symptoms of the cord or of cord and roots. It usually consists of an oval outgrowth of spongy bone with an irregular cortex and of about the same density as the vertebrae. The surface is capped irregularly by islands of cartilage which have the atypical structure of epiphyseal lines and through which growth of the exostosis occurs. Growth usually is most marked during the second decade and ceases about the age of 20 with the closure of the epiphyseal lines throughout the skeleton. Hence, symptoms usually begin during the second decade, although they may come on much later. Pain, except that arising from pressure, is not a feature of the disease and operative removal, if all of the cartilaginous covering is obtained, is not followed by recurrence.

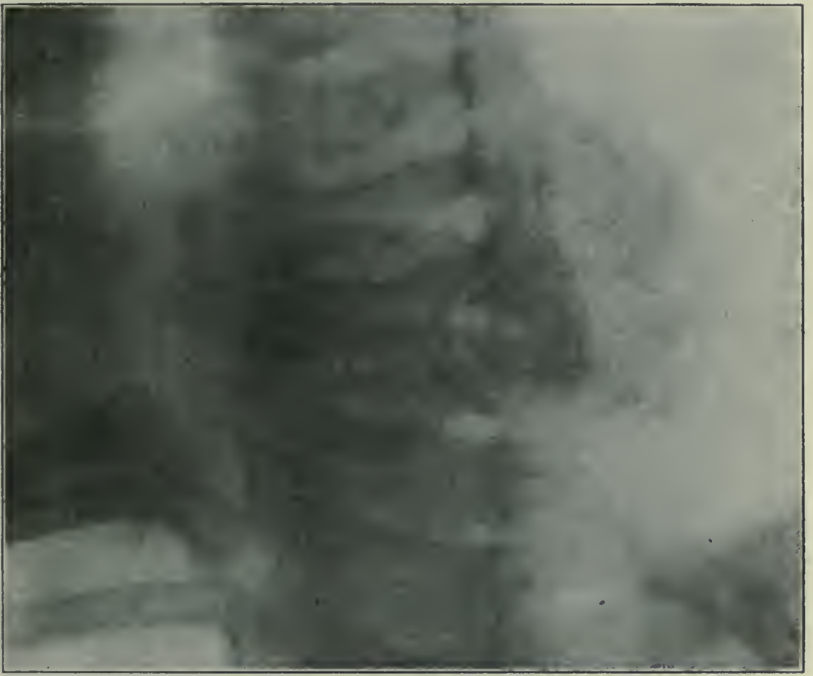

Fig. 1.—Exostosis of cervical region producing root symptoms.

Fibromas are sometimes seen, with root and cord symptoms, encroaching on the spinal canal and eroding the bodies or arches of the vertebrae. In such cases it may be difficult to say whether the tumor arose primarily from the bone or from the extradural structures of the spinal canal, but the occasional extensive erosion would seem to point to the bony origin. Bassoe reported such a case before this Society in 1915. The patient was successfully operated on by Dr. D. D. Lewis, first by a decompression operation in 1914 and subsequently by removal of the tumor in 1917. The patient has remained well to date.

Bone cysts, which are frequent in the long bones, have not been reported in connection with the spine.

Sarcomas show these variations from those of the other bones:

1. Marked ossifying forms are not so apt to occur.
2. Medullary giant celled tumors do not occur.
3. Hemangiomas and hemangiosarcomas are relatively common. About two thirds of such tumors affecting the bones have been located in the spine.
4. Mixed cell tumors are relatively frequent.

Their composition is variable, including myxofibrochondromatous and osteosarcomatous elements. Recently I have seen a more malignant form of the slightly calcified and ossified type of chondrosarcoma of the body of the fifth cervical vertebra, making root symptoms of one and one-half years' and cord symptoms of six months' standing. The destruction of the body, with isolated shadows scattered irregularly throughout the tumor, as shown by the roentgen ray, made the diagnosis very probable before confirmation by operation and pathologic examination of the tissue.

Fig. 2.—Mixed cell sarcoma from periphery of third thoracic vertebra making root symptoms.

Figure 2 shows a more benign form of this type arising from the side of the third thoracic vertebra in a woman, aged 36 years, who had suffered from left intercostal neuralgia for five years and from pain in the tumor region during the last two years. It was partly removed and treated by radium by Dr. D. D. Lewis. Histologically, it was a fibrosarcoma with extensive degeneration and isolated areas of cartilage and bone.

The commonest form is the round or spindle celled sarcoma which gradually destroys the bone and shows no tendency to ossify. Spine, root and cord symptoms are all liable to occur. Unless early death results from injury of the cord, the process is apt to extend through the periphery of the bone and make a tumor of considerable dimensions which facilitates the diagnosis.

A round celled sarcoma of the third cervical vertebra in a man, aged 38 years, had eroded the bone and led to marked pain with rapidly developing cord symptoms ending in complete paralysis below the level within two months of the onset. Pathologic fracture or dislocation may occur.

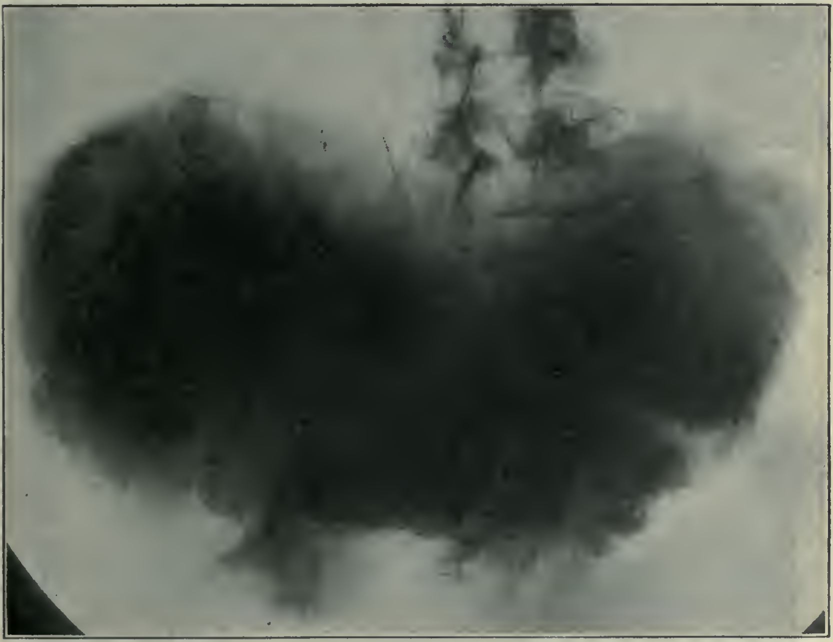

Fig. 3.—Spindle cell sarcoma of sacrum producing root symptoms. All of heavy shadow is tumor mass.

Figure 3 is a roentgenogram of a necropsy specimen of spindle celled sarcoma of the sacrum in a woman aged 28 years. It was of five years' standing and reached enormous proportions before death. There was no ossification in the tumor. The sacro-iliac joint was markedly displaced outward, and the fifth lumbar vertebra partly eroded. Intervertebral disks were not destroyed. There were marked local spinal symptoms throughout and a varying double sciatica during the first three years, after which motor symptoms developed in the limbs with disturbances of the bladder and rectal control, along with

Fig. 4.—Osteoclastic carcinoma metastasis from thyroid spine symptoms without collapse of body of first dorsal vertebra.

Fig. 5.—Metastatic osteoclastic carcinoma from breast collapsing second lumbar vertebra.

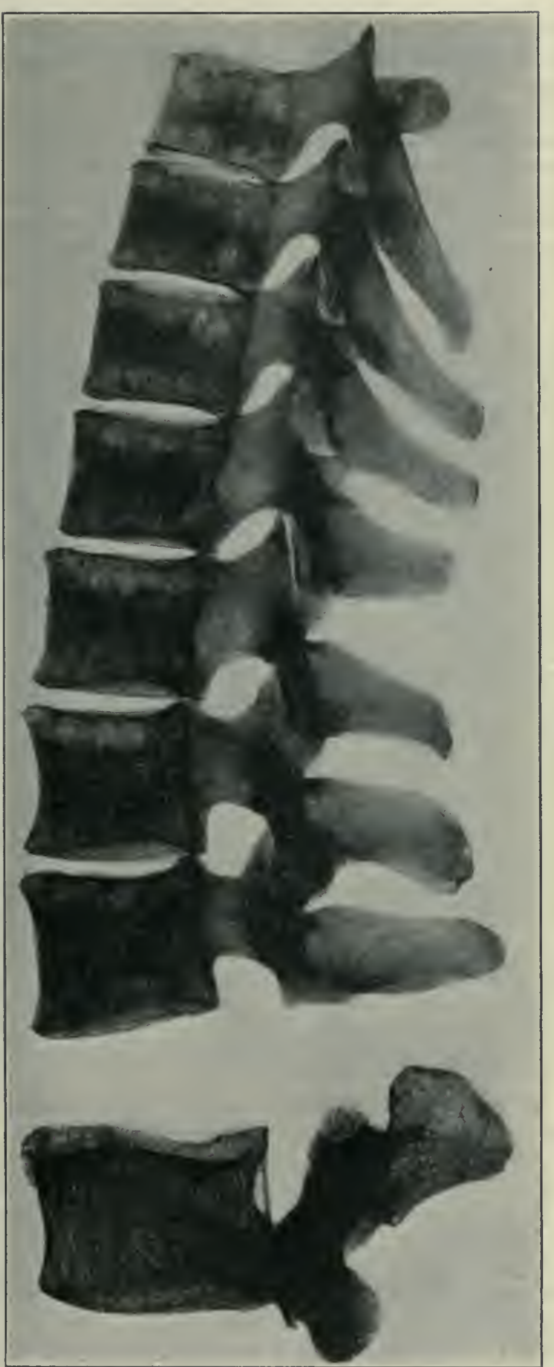

Fig. 6.—Roentgenogram of one half of bodies of thoracic vertebrae removed in case of carcinoma of prostate, showing osteoplastic metastases; below, normal vertebra for comparison.

a palpable tumor. The case was studied by Dr. Bassoe, and a lumbar exploratory operation performed two years after the onset of symptoms, with negative findings.

Metastatic carcinoma is by far the commonest tumor of the spine and is especially frequent when the primary carcinoma is in the breast, thyroid, kidney or prostate. There are two types with reference to bone destruction and new bone formation—the osteoclastic and the osteoplastic. The osteoclastic, or bone destroying form, is common, especially from primary tumors of the breast, thyroid and kidney. The lesions are frequently multiple and may be in any portion of the spine, although the lumbar region is relatively more often affected. Spine symptoms, especially a dull pain, frequently preceded the root and cord symptoms as in the case illustrated in Figure 4 of metastases in the first lumbar vertebra from carcinoma of the thyroid. Extensive metastases destroy the bodies of the vertebrae, leading to collapse, as shown in Figure 5, from carcinoma of the breast. The tumor frequently breaks through into the spinal canal, causing marked root and cord symptoms, and frequently to the outside with the formation of a mass about the bodies. Occasionally a small amount of ossification will occur in its substance.

Osteoplastic metastases are rarer and much less understood. They arise especially from carcinoma of the prostate (Figure 6), but occasionally from primary tumor of other organs, such as the breast. The metastases are usually small and diffuse, leading to marked new bone formation, filling out most of the cancellous spaces and giving the bodies and arches a dense structure which casts a heavy shadow in the roentgenogram. Breaking through the cortex rarely occurs, hence cord and root symptoms and shadows on the outside of the bodies are rarely present. Marked spine symptoms are common, consisting of shifting rheumatic pains similar to those produced by like metastases in the bones of the extremities.

Dr. Phemister, in closing, said that myelomas of the spine nearly always cause breaking down of the body, and symptoms corresponding to carcinoma.

Concerning the roentgen-ray treatment of cases of the destructive type, Dr. Pfahler of the University of Pennsylvania, showed at the American Roentgen-Ray Society a series of roentgenograms before and after roentgen-ray treatment, and it was astonishing to see how he could bring about an ossification in a nonossified spine, and convert it into a spine much like the one he had passed around. He also relieved the symptoms so that the patients lived for seven years. It was a most striking palliative therapeutic result from the use of radiotherapy.

Dr. Carl B. Davis: As an adjunct to Dr. Phemister's paper, Dr. Bassoe suggested that we report an interesting case which we have seen together recently.

The patient, a young woman, aged 28, reported to Dr. Bassoe with a history of pain on the inner aspect of the right arm and running around the chest in the region of the second and third intercostal spaces. The pain was worse when using the arm. When the shoulder girdle was at rest there was relief. There was no change in tactile, pain or heat sensations and no atrophy. There seemed to be diminished power in the arm.

Roentgen-ray examination showed a tumor mass involving the second, third and fourth ribs on the right side. The shadow extended out along the ribs for a distance of approximately 3 inches. The plates also showed osteophytes in the region of the wrist and knee joint. The osteophytes extended downward along the tibia and upward on the femur from the region of the lower epiphysis. Physical examination showed that she was normal otherwise.

Under general anesthesia the muscles attached to the right upper six dorsal spinous processes were freed and turned outward. The erector spinae muscle was pulled medianward. The tumor, including 2 inches of the second, third and fourth ribs, was resected. It had been hoped that a retropleural resection would be possible. At operation, however, it was found that the pleura was tightly adherent over the anterior surface of the tumor so that it was necessary to make a defect in the upper chest wall approximately 4 inches long and 3 inches wide. After removal of the tumor the right lung was seen in collapse. There was no shock and no pulse change other than a slight increase in rate. The muscles were stitched closely over the pleural opening in such a manner as to produce an air-tight cavity. The wound was closed without drainage.

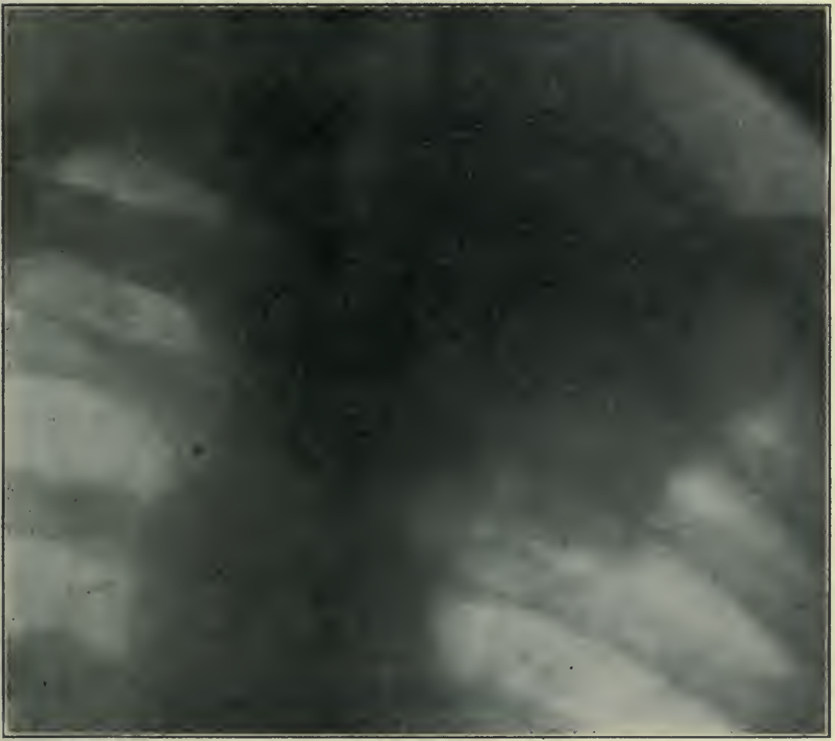

Fig. 1.—Tumor mass involving the second, third and fourth ribs.

The patient experienced an average convalescence until the fifth day. During these five days there was a slight elevation of temperature, a moderate increase in pulse rate and increase in the leukocyte count. On the morning of the fifth day while sitting up to eat her breakfast, she gave a gasp, and with all the symptoms of a pulmonary embolism died within a few minutes. Necropsy was refused.

The tumor proved to be a mass of bone and cartilage, probably an overgrowth analogous to the osteophytes that were found in the region of the epiphyseal line, due to abnormal development and closure in the epiphyseal lines.

The case was especially interesting because of the resemblance of the symptomatology to that of a cervical rib.

The second case is that of a patient who complained of pain in the neck and arm, made worse by carrying any weight in the hand on the affected side. Weakness and atrophy of the muscles in the ulnar region were present. The patient presented symptoms that might be present with a cervical rib. Roentgen-ray examination showed an osteoma arising from several of the lower cervical vertebrae. The tumor mass extended out into the neck in such a manner as to involve the roots of the brachial plexus. The mass was removed and the patient obtained relief for approximately two years. Recently there has been a return of symptoms and roentgen-ray plates show a recurrence of the tumor, larger than before. The cervical roots are involved in such a manner that there is now ulnar paralysis and atrophy with sensory changes in the ulnar distribution.

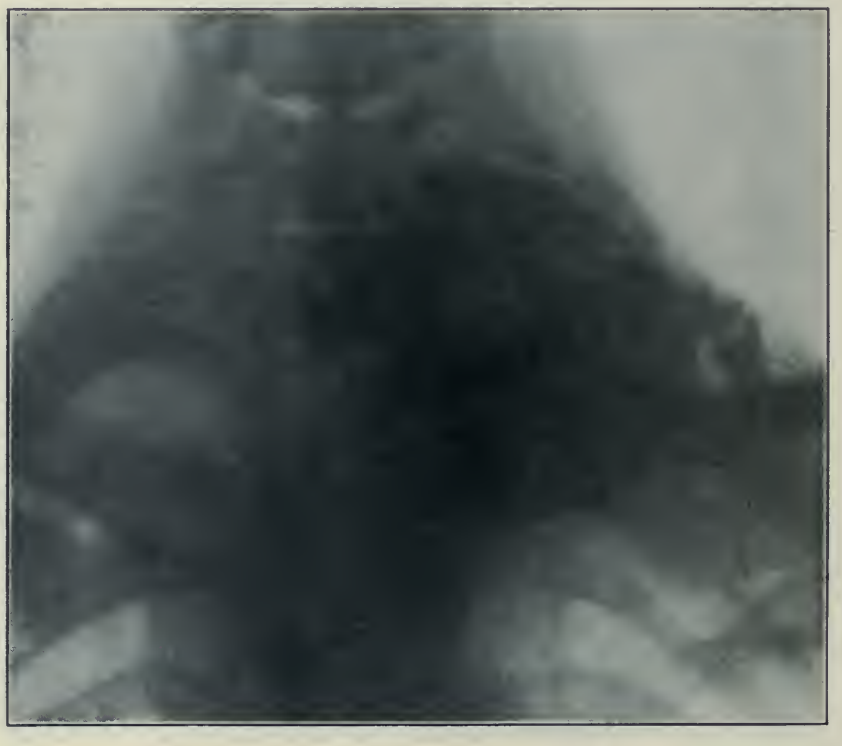

Fig. 2.—Osteoma in the cervical region.

DISCUSSION

Dr. Peter Bassoe said that one case of particular interest to him was that of a woman, aged 28, who complained of pain in the lumbar and sacral regions, and who was first seen in the summer of 1917. There was pain in the legs, more pronounced in the left; she had not walked alone for over two months.

He made a diagnosis of a lesion of the cauda equina. There were diminished pain and temperature sense in both legs, corresponding to the lower lumbar and upper sacral segments, atrophy of the leg muscles and absence of ankle reflexes. The Wassermann reaction was negative for both blood and spinal fluid; there was no increase in the cells. There were a strongly positive Nonne reaction, and a positive Lange test of a compression type. At that time nothing was found to be wrong with the bones on external or roentgen-ray examination. Dr. Dean D. Lewis performed a laminectomy, but found nothing except some small spots in the meninges. The patient went home, and a year later developed palpable and visible lumps about the pelvis. These had appeared six months after the operation. The lumps were large, firm, subcutaneous; the largest was 6 inches in diameter. The patient died about three months later, and the whole pelvis was removed at necropsy. There was a large, comparatively soft tumor all around the pelvis, filling the sacral fossa; there were also large tumors projecting backward, and further study revealed the fact that it involved the bone of the sacrum, and that it was a sarcoma. The interesting point was that the patient had had a sarcoma there all the time, and it had not produced any bone deformity, and it was two years after the cauda equina symptoms appeared that the presence of the tumor was recognized.

Other cases were less obscure. One woman, seen first in January, 1917, had pain in the hip, then in the buttocks and left leg; later in the right leg, more severe than in the left. Roentgenographic examination disclosed a bone defect in the iliac crest. Necropsy revealed a sarcoma.

The speaker had also looked up cases of carcinoma that he had seen (one person's experience did not amount to anything), but he had found eleven cases of primary carcinoma of the breast with metastases in the vertebrae, and three or four of primary tumor of the prostate, and two or three of uncertain origin. Some features about the breast cases were rather interesting. Two of these patients had been treated as neurasthenics. In one woman he found a very tender spine and a lump in the breast which no one had noticed before. He had the same experience with another patient. Women often concealed the presence of a breast tumor, but it was rather strange to have it happen when severe symptoms of metastatic growth had been presented for some time. In two of the cases abscess of the breast had preceded carcinoma. One patient had returned to the hospital where she had been treated for the breast tumor, but it was not noticed that there was a lump at the bottom of the scar of the abscess. Roentgenographic examination revealed extensive decalcification of the spine in several places. In one case, there had been an abscess in 1902, a tumor appeared in 1903, and the breast had been amputated in 1910, seven years after the tumor was first felt. The same year, pain began in the back and later in the legs, and the patient succumbed one and one-half years after the onset of the spinal tumor. In that case also there were metastases in the skull. In one case, the breast was removed and the patient complained of severe pain at the time of operation; the tumor was discovered only a few months before. In that case the only metastatic tumor in the body was in the vertebrae.

In one case of carcinoma of the breast, the tumor was found in 1910, removed in 1913, and there were no symptoms until 1916, when pain in the back and unsteadiness in the gait occurred. It was said by those who performed the necropsy that no lesion in the spine was found. The roentgen-ray disclosed nothing, but there were growths in the cord itself along the meninges. The posterior part of the cord was most affected, which was in accord with the cases reported by Dr. Phemister.

The earliest symptom in all the cases was severe pain in the back, with exquisite tenderness. In one case this was localized along the fifth rib. Backache and tenderness seemed to be the most prominent early symptoms, while symptoms of the motor roots had not been observed.

He had seen one rather peculiar case of bilateral sciatica in which the patient had had a tumor of the nose eleven years before, which had been removed. Dr. Hyde had said it was benign and there was no further danger, but nine years later there was a local recurrence, which was treated with roentgen rays; two years later the patient developed severe sciatica, first on one side and then on the other. When he first saw the patient he thought there was no tumor, but necropsy revealed a carcinoma of the vertebra. The interesting point was the unusually long latency, and the slowness in the development of the definite signs of the existence of tumor.

Dr. George B. Hassin asked what Dr. Phemister's experience had been with myelomas.

A CASE OF OCCLUSION OF THE RIGHT POSTERIOR INFERIOR CEREBELLAR ARTERY. Presented by Dr. George W. Hall.

A man, about 35 years of age, while on his way to work, suddenly became dizzy and fell to the ground, but did not lose consciousness. Examination at that time showed horizontal nystagmus and ataxia of the right hand and leg. He was hoarse for four or five days, and the pulse was slow for several days. One week later while walking on the sidewalk he had a tendency to fall to the right. He partially recovered, but had a recurrence of symptoms about one month later. At that time Dr. Hall examined him. His head was tilted toward the right, both pupils reacted to light and accommodation. The right pupil was smaller, the palpebral fissure was narrower on the right and exophthalmus was present. Optic disks were normal; hearing was normal. Facial muscles on the right side were paretic, and there was slight atrophy of the right side of the tongue. Hemi-anesthesia of the right side of the face and loss of pain and temperature sense over left side of the body were present. Tactile and position sense everywhere were normal. Dr. Hall thought that in many instances it was impossible to differentiate clinically between occlusion of the inferior cerebellar artery and the vertebral.

CHICAGO NEUROLOGICAL SOCIETY

Meeting of Dec. 18, 1919

Lewis J. Pollock, M.D., *President*

TUMOR OF SELLA TURCICA WITH PRESENTATION OF SPECIMEN. Presented by Dr. Ralph C. Hamill.

The patient was a man, aged 25 years, who died Oct. 20, 1919. He entered Wesley Hospital, Aug. 30, 1919, complaining of severe headache from which he suffered for seven weeks in 1914. At that time the examining physician said the headaches were due to heart disease. The attack that brought him into the hospital had lasted twelve weeks, during which time he had had very severe right-sided headaches at short intervals. The pain would last for from two to ten minutes and reappear every half hour. The attacks were precipitated by change of posture, especially sitting up, and were accompanied by vomiting and dizziness. He also complained of attacks of breathlessness and palpitation on exertion. Twelve weeks before entering the hospital, he had an epileptiform seizure and subsequently had four others, two of which were observed in the hospital. They all occurred on changing position, principally on sitting up, and lasted for two or three minutes, during which time he became more or less rigid; the pupils were dilated, the pulse about 50, and respiration stertorous. Inquiry failed to reveal any aura.

The past history revealed nothing of interest, except that at the age of 13 he fell on the ice, striking the right temporal region. He did not become unconscious and walked home, but said that his right eye had turned in ever since. Paralysis of the abducens nerve was noticed on entrance to the hospital. The family and personal history were negative, aside from the above.

Examination showed that the heart was enlarged, with a loud, systolic blow at the apex, a diastolic blow over the base and no accentuation of the pulmonic second. There were skipped beats which were very irregular in phase —might skip every third or tenth beat. Heart block was suspected.

Neurological Examination.—There was internal rotation of the right eye which could not be turned outward beyond the midline. The pupillary reflexes were normal and vision of the right eye greatly reduced but he could read with the left. There was no hemianopsia. Both disks were choked, the right one grayish-white, the outline was blurred and the vessels atrophic. Outlines of the left disk were blurred and there were some small hemorrhages near the disk. Hearing was normal. The ears being douched with cold water (two examinations) showed that the left reacted well, with nystagmus, dizziness and past pointing. The right reacted only at the expiration of fifty seconds. Sense of smell was normal. The gait showed nothing. The face had rather a blank look, but facial movements were normal. The tongue was in the midline. At times there was tactile anesthesia of the right cornea, which gradually increased during his stay in the hospital.

Strength of hands and arms seemed normal; he could supinate and pronate the hands equally on both sides, the right slightly slower than the left. The finger to nose and finger to finger tests were normal. There was no Babinski sign; Gordon and Oppenheim signs were occasionally dorsal in type, and pinching one calf often caused withdrawal of the other leg. The deep reflexes and the abdominal were normal, the cremasteric was diminished.

The spinal fluid was clear, with 4 cells; Wassermann and Nonne reactions were negative. The blood Wassermann reaction was also negative.

Tumor of the base was diagnosed, either in the region of the sella or in the posterior fossa. Radiographic examination showed a distinct change in the posterior clinoid processes; otherwise normal. On the day before operation upward movements of the eyes, of a nystagmoid nature, were present. The right ear still showed diminished reaction to cold. Influenced by the ocular movements and by the symptoms from the vestibular apparatus, suboccipital decompression and exploration of the right posterior fossa were advised. This was done. No tumor was found. The patient died from respiratory failure just as the wound was being closed.

Necropsy Examination.—This revealed a large tumor in the region of the sella turcica, which proved to be an endothelioma. The flattened hypophysis was found in the sella. The tumor had apparently started above the optic chiasm somewhere in the floor of the third ventricle; it was impossible to determine exactly where. The tumor surrounded and included the chiasm in its mass. The floor of the ventricle was pushed upward by the mass and also by a large cyst that appeared to lie on top of the solid tumor. The walls of this cyst paralleled the floor of the ventricle, giving it the appearance of having a false bottom. The posterior end of the cyst was very close to the opening of the aqueduct and doubtless caused a change in the physical conditions thereof. A notable symptom throughout his stay in the hospital was severe headache about three minutes after he changed from a lying to a sitting posture.

Weisenburg in Brain, 1910, divided tumors of the third ventricle into those which obstruct and those which do not obstruct the foramena of Monro or the aqueduct of Sylvius. In Class 3—those which grow into the aqueduct—paralysis of ocular movements, especially upward movements, is mentioned as a common symptom. The cyst in my case probably pressed on the upper end of the midbrain and the nystagmoid movements upward appeared as symptomatic of this irritation.

In tumors in the region of the floor of the third ventricle, as in the cases reported by Dr. Bassoe and Dr. Pollock, the tumors were in the ventricle rather than pushing the floor upward.

TWO CASES OF TUMOR OF THE HYPOPHYSIS. Presented by Dr. Burrell O. Raulston.

Case 1.—*Adenoma of the Hypophysis with Acromegaly.*—A woman, aged 28, complained of aching pains in the occiput, over the left eye, and a feeling of pressure back of the left eye. These symptoms had been present to some degree for about one year preceding September, 1919, during which time there had been noticed a progressive enlargement of the head, especially the lower jaw, and of the hands, feet and chest. The features had become decidedly coarser and vision had gradually failed, though she could still go about her house work. About one year previous to the above date, she had had three operations on her nose and accessory nasal sinuses within a period of three or four weeks. By one of these operations the left frontal sinus was opened externally. For a period of three weeks following this the frontal pains disappeared and there was a loss of the sense of smell.

The family history contained nothing of importance in connection with her complaint. She had two children, 3 and 5 years old, who were healthy and apparently normal.

The patient was of broad, heavy build with pale thick skin and coarse features. She presented the typical changes found in acromegaly of mild degree. The pupils were unequal and sluggish in their reaction to light and accommodation. A finger could be distinguished by the right eye at a distance of 6 feet, by the left, 8 feet. The right optic disk was pale from secondary atrophy, and in the left there were signs of beginning atrophy. The tonsils had been removed. A careful examination of the nose and nasopharynx revealed no growths. A shadow was cast by each frontal sinus. No abnormalities of the chest or abdomen were found, aside from the size and shape of the chest. Examination of the blood and urine revealed no noteworthy abnormalities. Systolic blood pressure was 122, diastolic 80.

Three hundred grams of glucose given by mouth at 7 a. m. failed to produce glucose in any one of hourly specimens during the five succeeding hours.

A diagnosis of tumor of the hypophysis and acromegaly was made. On Sept. 23, 1919, Dr. Dean Lewis attempted to remove a portion of the hypophysis by the nasopharyngeal route. When the floor of the anterior cerebral fossa was perforated it was thought that a large pulsating tumor mass could be felt in the region of the hypophysis. The wound was closed without removing any part of the tumor and the patient died about twenty-five hours later. Previous to the operation the patient's temperature had not been above 99 degrees per os, but shortly before death it reached 106 degrees per rectum.

At necropsy there was found a large bluish pedunculated tumor in the region of the hypophysis, marked flattening and symmetrical enlargement of the sella turcica. Marked increase of intracranial pressure was indicated by flattened gyri and shallow sulci over the cerebral cortex, and a foramen magnum furrow of the cerebellum.

The tumor mass extended from one temporal lobe to the other, a distance of 6 cm., from the anterior border of the pons forward against the frontal lobes, a distance of 4 cm. The tumor projected 2 cm. beyond the level of the frontal lobes. The optic chiasma was between its anterior surface and the frontal lobes of the brain. Each optic nerve was represented by a thin ribbon-like strand of tissue. When the tumor was pushed downward and to the right an irregular nodular portion was seen projecting between the left temporal and frontal lobes.

In histologic sections the predominating type of tissue was necrotic hemorrhagic material. In those regions that were lightest in color as seen macroscopically there were strands and groups of cells that resembled hypophyseal tissue closely and from which a diagnosis of adenoma of the anterior lobe of the hypophysis was made.

Case 2.—*Mixed Cell (Erdheim) Tumor of the Hypophysis.*—A woman, aged 57 years, first seen Feb. 12, 1919, complained of abdominal pain, painful urination, incontinence of urine and vomiting. The pain was cramp-like in the lower left quadrant of the abdomen, present for periods of about one hour at irregular intervals and was usually relieved by a bowel movement. The vomiting had been present about one week and occurred soon after meals. Urination had been painful for about one year; there had been incontinence two or three weeks. For thirteen years she had suffered from what had been diagnosed as gallstone colic, but she had had none of these attacks for three years. She had suffered with shooting pains in the legs for many years, but did not have any feeling of constriction about the trunk. Headaches had been frequent for several years and vision had gradually failed until she became totally blind three months previous to entering the hospital. Poor appetite, dyspnea on exertion and slight cough were also complained of.

The patient was a very obese elderly white woman, complaining of but little pain, but regurgitating brownish fluid frequently, and belching constantly. Vomiting was without distress. She drank frequently but had no desire for food. The pupils were large, round, equal, inactive, the lenses sclerosed and primary optic atrophy definite in each eye.

Further examination of the head and neck revealed no other abnormalities. The heart was thought to be enlarged, though no other definite changes could be determined. Subcrepitant and coarse râles were heard over the entire chest. While the abdomen was somewhat tender in all parts, no rigidity was present nor could any mass be palpated.

Wrist reflexes were not obtained on either side; the elbow reflexes were active, but the abdominal patellar and Achilles' reflexes were absent. The Babinski sign was present on the right side, questionable on the left.

There was a small amount of albumin in the urine, and a leukocyte count of 18,000. The temperature on admission was 100 degrees and it rose to 104 degrees on the second day, when she died. No spinal puncture was done, no blood Wassermann test was made.

The diagnosis was bronchopneumonia (?); syphilis; tabes dorsalis; primary optic atrophy; blindness; chronic myocarditis; ventral hernia.

Necropsy was permitted and the following anatomic diagnosis made: bronchopneumonia; fatty changes of the myocardium; marked hypertrophy and dilatation of the urinary bladder; chronic cholecystitis; cholelithiasis—stone impacted in the cystic duct; tumor of the hypophysis; marked atrophy of the optic nerves; petechial hemorrhages in the renal pelvis, the urinary bladder and the stomach; mild chronic diffuse nephritis; fat infiltration of the renal pelvis; senile sclerosis of the aorta and its main branches; marginal emphysema; moderate obesity; large ventral hernia; healed bilateral fibrous pleuritis.

A study of the brain after hardening in formaldehyd revealed flattened gyri and shallow sulci, especially over the superior and lateral surfaces of the cortex. The olfactory nerves were in their distal half in normal position and were apparently unchanged, but in their proximal half they were pushed laterally and compressed by a tumor mass which projected forward from the region immediately in front of the pons. The optic nerves were closely adherent to and on first inspection appeared to emerge from the sides of this tumor 2 cm. posterior to its anterior margin. The oculomotor nerves were in normal position and extended across the posterior surfaces of the tumor. The other cranial nerves as seen on the brain after removal were in normal position and presented no gross abnormalities. The tumor mentioned above extended from one temporal lobe to the other, with a maximum transverse diameter of 3.5 cm. and from the anterior margin of the pons forward for a distance of 5.5 cm. The visible portion of its surface was irregular in outline and consistency. Some portions had a light bluish color and were covered with a thin membrane, fluctuate, and were found to contain clear fluid. These cystic regions constituted from one-third to one-fourth of the visible surface and the remainder of the mass was about the color of brain tissue, but somewhat more firm. On the anterior superior surface of the tumor, when it was pushed away from the frontal lobes there was a cystic portion that had an anteroposterior diameter of 2 cm. When the brain and tumor were incised transversely through the region of the infundibulum the greatest vertical diameter of the tumor was found to be 3.25 cm. The surfaces made by sectioning the tumor were for the greater part made up of gray, granular material. There were small, irregular round regions of homogeneous, opaque, gelatinous substances, some of which represented coagulated fluid of cysts; others were not surrounded by a membrane. No regions were found that had the firmness of cartilage, nor was any calcium salt found.

On the left lateral wall of the tumor beginning 2.5 cm. posterior to its anterior border there was an oblong brown and yellow body that measured 17 mm. longitudinally, 5 mm. transversely and 8 mm. vertically. Surfaces made by sectioning the body presented solid nongranular yellow and brown tissue arranged in concentric striations about an upper and lower center.

Histologically the tumor contained regions of deeply staining epithelial cells that were irregular in shape and size. Each of these regions was surrounded by a more heavily stained single layer of columnar epithelium. Many cysts were seen. They were likewise surrounded by a single layer of epithelium and contained masses of poorly staining homogeneous substance. An average of about one third of each microscopic field was made up of squamous epithelium that had undergone extensive hyalin degeneration and stained poorly. This tissue was very compact and in most places many layers thick. The nuclei stained poorly and in many places could not be made out at all. There was a large amount of necrotic substance that was in most places surrounded by a vascular connective tissue or columnar epithelium. No cartilage cells were present.

The histology of this tumor conformed to the description of those said to arise from inclusions of the craniopharyngeal duct and described first by Erdheim. The mass was located in the region of the hypophysis and since this gland could not be found it appeared that the tumor arose from inclusions of the above named duct in the anterior lobe of the hypophysis.

DISCUSSION

Dr. Peter Bassoe was interested in Dr. Raulston's remark that in the second case he reported an erroneous diagnosis of tabes was made. There was nothing new about such errors and Oppenheim wrote a paper on this subject some nine years ago (Oppenheim, Ztschr. f. d. ges. Neurol. u. Psychiat. **25**:527, 1914). Clinically, the case seemed quite clearly to be one of tabes and yet no tabes was present but a tumor of the hypophysis, and he confessed that he had made that mistake before.

Dr. Clarence Neymann called attention to some serologic findings in brain tumors which might be of assistance. The Pandy test for globulin in the spinal fluid was not absolute in general use, but was of value in brain tumors as well as in epilepsy. In both diseases, one often found a negative Ross-Jones test and a positive Pandy reaction. The colloidal gold curve obtained in spinal fluids of brain tumor cases very nearly approached that of tabes. He considered these two tests of value in the presence of the Ross-Jones and Wassermann reactions and as being somewhat indicative of such organic brain lesions. The Pandy reaction was a sensitive reaction, and he thought should be employed more than at present.

Dr. Ralph Hamill said that when he was at Dunning he had a case of tumor of the pineal gland with marked ataxia. At postmortem examination the posterior columns were found to stain with osmic acid and also showed some degeneration with Weigert-Pal. There was no evidence of syphilis in the membranes of the spinal cord. To strengthen the supposition that it was syphilitic, about a week before the patient died she had a diplopia which lasted for a day or two and was probably due to a displacement of the tumor process down on the oculomotor centers.

SAN FRANCISCO NEUROLOGICAL SOCIETY

First Meeting, Dec. 5, 1919

Walter F. Schaller, M.D., *President*

SUMMARY OF OBSERVATIONS ON THE TREATMENT OF HYSTERIA WITH ESPECIAL REFERENCE TO RELAPSES. Presented by Dr. Julian M. Wolfsohn.

Detailed analysis of the literature and of the cases personally studied by the author has led him to believe that all war psychoneuroses could be segregated into three great groups: (1) hysteria, (2) neurasthenic and emotive states, (3) a combination of (1) and (2). Each of these three groups show one or all of the fundamental defense reactions of physical or psychic pain, fatigue and fear. Traumatic psychoneuroses resulting from injuries incurred in industrial accidents and those of civilian life often present the syndrome of war psychoneuroses in as complete a form as is seen in the pathology of war.

In the treatment of psychoneuroses, whether due to accidents of war or industry or to the defense reaction found in an intolerable environment in civilian life, should not all cases be susceptible to cure by the same means? Some critics say, "No"; that the psychoneurotics of civilian life are not amenable to the methods used in the army in which all patients are under the thumb of discipline and in which compensation, as such, is a negligible quantity. As a matter of fact, military, civilian and industrial cases of hysteria are cured of their symptoms in the same way, by reeducative methods, even after other long, previously unsuccessful, methods have been used

This report is based on 1,550 cases of psychoneuroses—type-conversion hysteria—treated and cured of their functional disabilities by the method described in a previous paper (J. A. M. A. **71**:2057 [Dec. 21] 1918). These cases represent: hysteria directly resulting from war activities, 1,500; traumatic hysteria of civilian life (industrial accidents), 10; conversion hysteria (emotional in origin) of civilian life, 40.

No patient was treated unless at least an "outward" desire for recovery was shown. This was the signal for a shorter or longer talk with the patient of a psychanalytic nature in which dreams were interpreted and complexes unearthed. It was at once seen that patients without much outward emotivity could be cured without extended psychanalyses, while those presenting evident emotivity as a symptom, required prolonged analyses. When there were many patients awaiting treatment, and at times there were from ten to twelve in a day, a glance at the table of relapses shows that it was impossible to make sufficiently detailed psychanalyses. Hence, patients with conversion hysteria associated with emotivity were prone to relapse when an unfavorable environment was encountered.

The results of treatment in a former series of 573 cases are merely accentuated by this series which is twice as large. One fact stands out boldly, however, and that is the great value of preparing the patient for cure by psychanalysis and dream analysis. This is necessary in every case in order to unfold the causative psychic factor. The difficulty of treatment with repressions such as these is apparent when psychanalytic methods are ignored.

The author's interest was early aroused as to what percentage of his patients would relapse after the treatment and cure of the functional disability. For this reason, each patient was given, on discharge, two postals to the author—one dated three months and the other six months after the cure. Of the first 500 replies it was found that definite relapses occurred in 16 per cent., or eighty cases. Four hundred and twenty patients wrote that they had remained perfectly well three and six months after treatment although many of them had returned to the former environment.

The patients who suffered were: twelve mutes, seventeen aphonics, thirty-eight stammerers, twelve tremblers, one ticquer. In each one of these at the time of cure, emotivity evidenced by easy blushing, fears of various kinds, headache, insomnia, terrifying dreams, starting reflex, etc., was a part of the symptomatology of the disease. This residual emotivity when present was the cause of the relapse. Sudden shock, noises, crowds, bewilderment or even an environment in which fear plays an important part, may be the starting point of the relapse. Hysterical cases incurred in the army; in industrial life, in which compensation was a factor; and, finally, in civilian life where emotivity pure and simple was the cause, were described.

As vivid, terrifying dreams often cause the sudden cure of a disability—e. g., a mute will awaken to find himself shouting and will thereafter be able to speak—so the terrifying dream is also potent, when emotivity exists, as a factor in the relapse. Therefore, it was early seen that, in order to treat successfully for permanency of results, a course in mental catharsis was necessary to clear the psychic factor or minimize it and that it is the most helpful means at our disposal to prevent relapses.

After having seen psychoneuroses under the most varied guises, the author says one of the most important conclusions can be drawn—that hysteria of whatever cause has an essentially psychic character and gives origin exclusively to psychogenetic disorders and that, when a painstaking psychanalysis is made, suggestion is easier to administer and ultimate results are far happier.

DISCUSSION

Dr. Thomas G. Inman said that in his experience the question of cure in hysteria was a difficult, if not an impossible, one to decide. He had known somatic symptoms to remain absent for more than twenty years only to recur under the influence of a new emotional stress. There was much similarity between the patients observed as war cases and those seen at home, and he thought the mechanism was the same. After proper preparation a cure at one seance was thought to give the best results as regards economy of time, and it did not seem to make much difference what means was used so long as the suggestion was sufficiently firm. There seemed to be considerable difference in the intensity of the fixation in different persons, some recovering during the process of examination while others resisted the most extreme measures. Yet all are amenable to symptomatic cure by some method, the difficulty being in the selection of the proper one.

Dr. Harold W. Wright wished to express his agreement with Dr. Wolfsohn as to the value of psychanalysis in bringing about a fundamental cure of traumatic hysteria or other forms of psychoneurosis. However, the mental complex is not always so obscure or so deeply subconscious as many psychanalysts would have us believe. As to the war neuroses, he thought that all who were working in this field, especially overseas, agreed pretty well as to their nature and their mechanism; in fact, it was striking to see so little disagreement as the war progressed. Of course, in the rush of patients during the period of hostilities and the necessity of getting as many as possible of the men back to duty in a short time, it was not possible for those who were on duty in France to practice very thorough psychanalysis. One had to be content with removing symptoms first of all and this was found to be fairly easy. But it was always explained to the patient how the cure was produced in such a way as to convince him that he had not sustained any organic lesion, analyzing his case for him briefly in order to fortify him against relapse. Often it seemed essential to assure the patient first of all that he would not again be sent to the fighting zone after being cured, but would be given duty in the intermediate zone for which he would be quite fit. In this way 80 per cent. of our patients were made fit for some form of duty within an average period of three weeks. Those physicians who worked in the advance hospitals in France and received patients soon after onset were in an exceptionally favorable situation to obtain results because there had been no chance for the neurosis to become fixed or aggravated by delayed treatment or by unwise suggestions resulting from many examinations by various physicians who were not sure of their diagnosis. It is deplorable that the traumatic neuroses of civil life cannot be controlled as promptly and as efficiently.

SURGICAL EXPERIMENTS IN THE ARMY WITH ORGANIC LESIONS OF THE NERVOUS SYSTEM. Presented by Dr. Howard C. Naffziger.

Brain injuries are treated chiefly at the front in the farthest advanced hospitals. Peripheral nerve injuries are treated at the bases most remote from the front. With the former the most profitable observations have been made along physiologic and bacteriologic lines, i. e., the reaction of the central nervous system to trauma, hemorrhage and infection. With these gross lesions, detailed neurologic study is not practicable, necessary or profitable at the front. More is gained from later study when the complicating factors have been removed. The principles guiding surgery of the brain in war wounds are given.

Peripheral Nerve Injuries: The methods of examination and records are discussed. Emphasis is given to the necessity of recording voluntary power in the individual muscles. For each nerve a chart is shown which lists the muscles in the sequence in which they receive their nerve supply. Proper spaces are allowed for recording faradic and galvanic reaction of each muscle, on the same chart. Two examinations, one month apart, are considered necessary in order to form a proper estimate of the progress of a case.

Joint stiffness has been a most troublesome complication. Much emphasis has been placed on the necessity of using splints to prevent overstretching of paralyzed muscles. As a result, most of the patients have worn splints. Unfortunately, in many the mobility of the joints has been sacrificed. The parts have not been put through their full range of motion at sufficiently frequent intervals to preserve mobility. The improper use of splints has contributed greatly to the disability. Vasomotor, trophic changes and atrophies have been more pronounced when the part has not been used.

End to end suture of nerves with accurate approximation is considered the most favorable operative procedure. While "cable grafts," i. e., autografts of several strands of nerve from the same person, are used when end to end suture cannot be performed, it has rarely been necessary. With increased operative experience grafts are more and more rarely necessary. Mobilization of nerves, transposition and correct joint positions enable one to secure end to end suture in ulnar, musculospiral and sciatic nerves with gaps of 10 or more centimeters. Greater gaps can be overcome by two stage operations.

Technical details in methods of exposure, handling of nerves, examination of neuromas and nerve suture were presented and illustrated by drawings by Mr. Ralph Sweet. Two reels of moving pictures showing ulnar, median, musculospiral, external popliteal, sciatic, faciohypoglossal and facial paralyses were shown. In each of these the paralyzed extremity was compared with the normal one and methods of examination for individual voluntary muscle power were shown. Each part was put through the full range of all voluntary movements under examination and the characteristic attitudes, atrophies, gaits, etc., were shown.

DISCUSSION

Dr. Thomas G. Inman stated that frequent electrical examination gave the most trustworthy information as to the severity of a nerve injury in the absence of evidence as to the exact nature and extent of the injury. Rapidly diminishing galvanic irritability spoke for the separation of the continuity and operation should not be too long delayed. The moving pictures of nerve injuries and the disturbances in function associated with them were exceedingly clear and complete and would be invaluable for class instruction.

Dr. Harold W. Wright had the pleasure of seeing the first hundred of the series of cases at Letterman Hospital with Dr. Naffziger. He mentioned a few facts that seemed to be of great help in arriving at a conclusion as to the prognosis of unoperated cases and as to the decision whether or not to operate.

One of the most important is a tenderness of the muscles supplied by the affected nerve which is always present when the nerve is regenerating and which may be found before voluntary power is apparent. This sign, together with an advancing tingling on percussion, or Tinel's sign, seemed to him to be very assuring and this was borne out by subsequent examinations. As to the electrical reactions, he felt that they should not be taken too seriously in these peripheral nerve wounds because often reactions of a degenerative type have been observed when voluntary power is present in some of the muscles or has returned to them so that it would not be safe to advise operation solely on account of the electrical responses. Another point worth emphasizing is the importance of excluding compensatory action of other muscles physiologically related and this can be done only by testing each muscle carefully and securing a posture of the part tested which will prevent the action of normally acting muscles. This was well shown in Dr. Naffziger's pictures. It is easy to be misled in this matter, especially when testing the small muscles of the hand.

MINNESOTA NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 17, 1920

Charles R. Ball, M.D., *President*

A PROBABLE CASE OF MARIE'S HEREDITARY CEREBELLAR ATAXIA. Reported by Dr. C. E. Riggs.

From the "erratic" cases of Friedreich's ataxia, Marie, twenty-seven years ago formulated a syndrome which he believed differed both clinically and anatomically from that disease. In such cases, ataxia develops in the lower extremities after the twentieth year; several years later the arms are affected;

there are nystagmus, speech disturbances, optic atrophy and loss of pupillary reaction to light. The knee jerks may be normal or spastic; ankle clonus may be present; deformities of foot or spine are wanting; pain in the lower limbs and disturbances of sensibility occur; mental impairment has been observed.

My patient's syndrome may be considered "erratic" when contrasted with either Marie's hereditary cerebellar ataxia or Friedreich's disease. He was 24 years of age. The family history was negative. He had had the usual childhood infections. Unsteadiness in standing and walking began in the eighteenth year; the arms became affected four years later.

The pupils reacted normally to light and accommodation. There was no nystagmus. The eye grounds were normal. Speech was slow and hesitating. He experienced difficulty in uttering words and framing sentences. Knee and Achilles' reflexes were active and the Babinski sign was present on both sides. Ankle-clonus was present but of short duration. The superficial reflexes were normal; there was no disturbance of sensation. On losing his balance, he fell unless he was able to grasp something to support himself. When he tried to run his feet moved like flails and he did not move as rapidly as when walking; otherwise the result of the examination was negative.

We now believe that Marie's syndrome is a variety of heredo-degenerative disease of which the Friedreich syndrome is the most frequent expression; it has an endogenic basis and is characterized by steady progression. The observations of Bing and Thomas and Roux have made this clearly apparent. Clinically, we know that the Marie syndrome occurs in children as well as in adults; that the optic nerve and general sensibility may both be intact; that "the Friedreich foot" may occur; that generally the pupillary light reflex is absent, and finally that the normal or exaggerated knee jerk which has been commonly regarded as an absolute sign of distinction between these two conditions, may be increased or spastic at first and afterward lost during the course of the disease. Hence all the clinical and anatomic boundaries that have been supposed to exist between the Marie and Friedreich syndromes disappear, giving us a resulting simplicity of conception long sadly needed.

ATYPICAL JACKSONIAN EPILEPSY. Presented by DR. E. M. HAMMES.

A man, aged 27, was wounded June 7, 1918, in the left occipital region by shrapnel, but he continued to advance. About one hour later, he was wounded in the left arm and soon after a bullet passed through both thighs. He retreated and in about five hours (as far as he knows) became unconscious. He remained so for twelve days and in a semiconscious state for ten days longer. He then discovered that he was in a hospital near Paris, that he was unable to talk and that his right arm was paralyzed. By October, 1918, he had recovered completely, except for numbness in the palmar surface of his right hand which has remained. In November, 1918, he developed an attack that began with an increase in the numbness of his right hand which gradually extended up the arm to the shoulder where it stopped. About this time he developed motor aphasia. This condition gradually subsided after about one-half hour. He had three similar attacks, one in March, one in May and one in November, 1919. He has no other complaint.

The results of the neurologic examination were negative throughout. He had a superficial scalp scar over the left posterior occipital region. A roentgen-ray picture of the skull revealed a foreign body (probably steel) in the left occipital region and in the left upper parietal region immediately beneath the skull.

This patient probably had a subdural hemorrhage with subsequent adhesions.

HEMORRHAGE IN THE CAUDA SECONDARY TO LUMBAR PUNCTURE. Dr. E. M. Hammes reported the case.

A man, aged 34, with a negative family and personal history, developed epidemic cerebrospinal meningitis in March, 1918. He was given daily intraspinal injections of antimeningococcic serum and he improved after the first five injections. During the sixth lumbar puncture at the third lumbar interspace, before any serum had been injected (according to the patient's statement, no records are available) he suddenly developed a flaccid paralysis of both lower extremities with loss of sensation. He had loss of sphincter control and had to be catheterized for two weeks, after which he gradually improved. Dec. 15, 1919, he had entirely recovered except for attacks of weakness in the lower extremities after exertion, which lasted for several hours. He described the attacks thus: After working for about six hours (section hand) he notices a weakness in both lower extremities, which becomes more marked and in about one-half hour his knees suddenly give way and he falls. The muscle strength returns after about thirty minutes' rest, when he can again resume his work.

The results of the neurologic examination were negative, except that both knee jerks and Achilles' reflex were absent. There were no sensory disturbances or atrophies in the lower extremities.

A REPORT OF NINE CASES OF MENINGO-ENCEPHALITIS. Presented by Dr. C. E. Nixon.

These cases occurred in a village in southwestern Minnesota. Five occurred in two related families and there were five deaths. The first possible case was that of a youth, aged 19, who came home one night complaining of not feeling well. The following morning he was nauseated and "felt tired." At night he became restless and delirious with periods of unconsciousness, and he died the next morning. The second patient had been visiting in the village for three days, leaving the day after the death of the first patient. While on the train going home he became ill, and within six hours after the onset became unconscious and died in about twelve hours. The diagnosis was said to have been spotted fever. The cases following these two showed well marked meningeal symptoms. The spinal fluids, however, were not positive for the meningococcus. Necropsies of two showed well marked meningeal exudate; unfortunately, a microscopic examination of the brains was not made. Most of the patients had well marked cranial nerve involvement. The last case was that of a boy, aged 12, who became ill twenty days after the first patient. For seven days of his illness, he showed no evidence of meningeal irritation, but had definite symptoms of encephalitis. Respiration was deep and rapid—from 40 to 60 a minute. He had diplopia, strabismus and nystagmus. He was semidelirious, muttering unintelligent phrases and picking at "objects" in the air. There was a twitching of the skin of the face. He complained of pain in the extremities, but was not sensitive to touch or pressure. He was definitely lethargic, and complained of feeling tired and drowsy. When aroused, he answered questions clearly, and memory was unimpaired. There was a coarse, jerking movement of the extremities, somewhat resembling chorea.

After the first week of his illness, he developed rigidity of the neck and a positive Kernig's sign. The cell count of several spinal fluid examinations was relatively low, and the meningococcus was not found by smears, inoculations or cultures. Microscopic examination of the patient's brain demonstrated perivascular infiltrations of lymphocytes and other changes in the brain stem and basal ganglions, commonly reported in epidemic encephalitis.

Book Reviews

DISEASES OF THE NERVOUS SYSTEM: A TEXTBOOK OF NEUROLOGY AND PSYCHIATRY. By Smith Ely Jelliffe, M.D., Ph.D., formerly Professor of Psychiatry, Fordham University, New York, and formerly Adjunct Professor of Diseases of the Mind and Nervous System, New York Post-Graduate Medical School and Hospital; and William A. White, M.D., Superintendent of St. Elizabeth's Hospital, Washington, D. C.; Professor of Nervous and Mental Diseases, George Washington University, and Lecturer on Psychiatry, U. S. Army and U. S. Navy Medical Schools. Third Edition, Revised, Rewritten and Enlarged. Cloth. Pp. 1018, illustrated with 470 engravings and 12 plates. Philadelphia and New York: Lea & Febiger, 1919.

A departure from orthodox classifications of nervous diseases must be viewed with considerable interest. In this volume the authors propose a classification in which the nervous system is divided into three levels of activity: the vegetative, the sensorimotor and the psychic. In the region of vegetative neurology, "a rich variety of disturbances is found, involving the glandular, gastro-intestinal, genito-urinary, vascular, respiratory, muscular, cutaneous and bony systems. In addition, there are certain complex clinical groups involving, for the most part, the glands of internal secretion, the endocrinopathies." The "field of sensorimotor neurology, including the disorders of the cranial and peripheral nerves, the spinal cord, the medulla, pons, brain stem, midbrain, cerebellum and cerebrum is that portion of neurology to which the term 'neurology' is generally thought of as applying to all intents and purposes, exclusively." The third and the highest level is the psychic.

The inclusion of disorders of the vegetative system in a textbook of nervous diseases is timely and proper. As to how much space should be allotted and how much importance be given them, doubtless opinions would differ. They fill 162 pages out of a total of 649 on descriptive neurology.

The first chapter is devoted to methods of examination of the nervous system. This is well presented and includes an adequate description of the examination of the vegetative system. Methods of mental examination are briefly sketched and several good questionnaires included. Electric examination is barely mentioned; no reference is made to technic or indications. There is throughout a strong tendency to refer to theories which are more or less controversial. In discussing organ inferiority (page 25) the authors refer to the recent observation, that, "in the ascendants of pellagrins there was a noticeable tendency to diseases of the skin and of the gastro-enteric tract. In the interpretation of such a fact the evident relationship of the skin, mucous membrane of the stomach and intestines has to be correlated with the presumed etiologic factor of the disease as worked out by Goldberger and his associates." Part I is devoted to vegetative or visceral neurology and the endocrinopathies and considerable controversial matter is included. Reference to the influence of psychoneuroses, hysterical conversions, compulsive tics and psychotic projections on pupillary inequalities and sympathetic paralysis, are a reflection of the authors' views on the influence of the unconscious and the importance of psychanalysis. A psychically determined erythromelalgia is mentioned. It is said that Raynaud's disease may be of psychic origin (shock, hysteria, schizophrenia), and the treatment of such an erythromelalgia is stated to be psychanalysis, and of Raynaud's disease, psychotherapy. In discussing the etiology of scleroderma the authors think that "inasmuch as many, if not most, hyperthyroidisms are distinctly psychogenic, at least in the initial stages, it is not an unreasonable hypothesis that psychogenic factors may form a part of the scleroderma pathogeny." In the chapter on the endocrinopathies are included such diseases as myasthenia gravis, myotonia congenita, myotonia atrophica and the myopathies.

Stronger evidence of the devotion of the authors to the psychanalytic school is found in their statement (page 283) that "unconscious sadistic states produce transitory and even chronic arthritic changes."

In the description of sensorimotor neurology an occasional contradictory statement is found. Although on page 249 it is stated that among the neighborhood symptoms of diseases of the hypophysis "the optic nerve changes, bitemporal hemianopsia, are the most important," on page 304 the authors say that "a lesion behind the chiasm, usually in the sella turcica, and not infrequent, as in pituitary disease, causes a binasal hemianopsia, partial or complete."

The authors say that "a true psychogenic tic douloureux is always a possibility," and "psychogenic cases which are numerous are curable by psychanalysis." This may be true of face pain. It certainly is not true of tic douloureux. The true significance of "dolorgenetic" (algigenetic) zones in tic douloureux is not properly interpreted and hypersensitive Valleix's points are confused with them.

One page is devoted to the infectious types of multiple neuritis, whereas about four pages are devoted to the thalamic syndrome. Notable, likewise, is the large amount of space devoted to sensory changes in cortical lesions—a highly controversial subject—in contrast to the small amount apportioned to the hemiplegic state. The statement that treatment by alcohol injections is without avail in causalgia is not in accordance with the reviewer's experience. Peripheral nerve injuries are well described. Some of the matter included, such as the classifications of Mme Dejerine and Mouzon, has already been discredited.

The lesions at the level of the medulla, pons, brain stem or midbrain are brilliantly described. The numerous illustrations from Dejerine help to make the chapters on localization in the brain and cord of great value. The cerebellar syndromes and the paralysis agitans group of diseases are treated well and in accordance with recent studies of these disorders. The chapter on neurosyphilis is very satisfactory.

The introduction to Part III, psychical or symbolic systems, is illuminating. From the student's standpoint it could, perhaps, be said that the various theories, other than Freud's, of the pathogenesis of the neuroses, should receive more liberal consideration. The symptomatology of hysteria is too briefly described.

The technic of psychanalysis is comprehensively presented. As most of the students of a textbook of neurology will not be able to put into practice, personally or vicariously, the method of psychanalysis, it seems to the reviewer that some space should have been allotted to the description of other methods of treatment which are successful in removing at least some of the symptoms of the neuroses. As could be expected, the authors devote considerable space to the consideration of the "meaning of the attack" in epilepsy, emphasizing the relation of the attack to a wish fulfilment, whereas practically nothing is said of other important theories of the pathogenesis of the epileptic convulsion.

The chapters on the psychoses present in an orderly, condensed, but comprehensive manner, the descriptions of the various insanities. They include the more recent views of the pathogenesis of some of the symptoms, which are presented in a clear and acceptable manner.

The volume is an important contribution to publications on neuropsychiatry. It contains within a small space a digest of much of the recent work on the unconscious, the vegetative system, the physiology of the nervous system and localization of lesions. It makes this matter accessible to some who could not otherwise become familiar with it. If the volume were intended only for the use of those familiar with neurology, it supplies a well-defined need. As a textbook for students and as a guide for the general practitioner its value is questionable. It contains a great amount of information and much that is most interesting but in its use the inexperienced student will need the constant assistance of a compent mentor.

NERVOUS AND MENTAL DISEASES. By Archibald Church, M.D., Professor of Nervous and Mental Diseases in Northwestern University Medical School, Chicago; and Frederick Peterson, M.D., formerly Professor of Psychiatry, Columbia University. Ninth edition, revised. Octavo volume of 949 pages, with 350 illustrations. Philadelphia and London: W. B. Saunders Company, 1919. Cloth, $7.50 net.

This being the ninth edition of what has proved to be a standard textbook, a detailed review is superfluous; especially, as the present edition differs but little from the preceding one, except that "the subjects of general paresis and traumatic insanity have been rewritten."

Avowedly the authors have in mind the immediate needs of the average medical student and general practitioner, and these needs are well met. Both authors have the art of simple and clear statement, they avoid hypotheses and controversial matter and they show excellent discrimination between the essential and nonessential.

In the department of nervous diseases one might desire a little more careful revision. Somewhat at random we note: that in the treatment of trifacial neuralgia, much of the matter is antiquated and some inaccurate—e. g., the statement that Spiller and Frazier divide "the sensory root above the ganglion but outside the skull;" that the distinction between tic and spasm is not clearly drawn; that the essentially toxic origin of angioneurotic edema is scarcely indicated. In a few places the bibliographic references and statements of priority are inaccurate.

On the other hand, many chapters are models for the end in view. We may instance those on arterial brain diseases and on tumors of the brain, and there is an excellent table of the symptoms caused by a cord lesion at different levels.

The 231 pages on mental diseases contain about as good an exposition of the subject as could be presented within such limits. But we suspect that the authors' personal predilections have had something to do with the relative space given to different subjects. Why should a brief textbook for students and general practitioners be lumbered with twenty pages on craniometry, palates, ears, etc., and only the same number given to manic-depressive insanity including involutional melancholia? Why should idiocy fill more than twice as many pages as dementia praecox and general paresis combined? But the modified table of Preyer on the steps of development of the normal child is excellent for a textbook and too rarely included.

The printing is good, the illustrations excellent and the index complete.

THE DIAGNOSIS OF NERVOUS DISEASES. By SIR JAMES PURVES STEWART, K.C.M.G., C.B., M.D., EDIN., F.R.C.P. 557 pages. Edward Arnold, London. 1920. Fifth edition.

The fact that this book has reached the fifth edition is sufficient evidence of its usefulness and popularity. While the present edition has been revised and partly rewritten, there is practically no change in its makeup. An additional chapter has been added on the war neuroses regarded from the clinical standpoint; no attempt is made in this chapter of about twenty-two pages to discuss theories. The discussion on the organic war lesions is continued and is a valuable addition to war neurology.

The other parts of the book have been revised here and there; some illustrations have been added and these are uniformly excellent. The chapters on peripheral nerve conditions have been revised and are excellent. Perhaps only in the discussion of cerebellar diseases has advantage not been taken of recent ideas on cerebellar localization and function. There is, however, very little fault to find, for Stewart's diagnosis of nervous diseases is too well and favorably known to merit other than favorable criticism.

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ENDOCRINOLOGIC ASPECTS OF SOME NEUROLOGIC CONDITIONS *

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NEW YORK

Just forty years ago, in 1880, there appeared an article on myxedema in the *Boston Medical and Surgical Journal* by Dr. William A. Hammond. He reported the first case of the kind in America. Based on necropsies of similar cases in England, he came to the conclusion that the mental symptoms were due to the mucoid deposits throughout the brain structure and that probably the cause of the disease lay there. No mention or thought of thyroid disturbance entered. Since that time we have made some progress. Does any one doubt that today, confronting us, are states of disease in which we show the same lamentable lack of knowledge? In the same year, Dr. George M. Beard presented at the American Neurological Association a report on neurasthenia—nerve exhaustion—with remarks on treatment; Dr. Hammond presented a paper on thalamic epilepsy, and Dr. Gray a paper on the "Interconvertibility of Migraine and Epilepsy." The juxtaposition of these papers gave me a text for this evening's address, made forty years after their presentation. The only one of the affections cited, whose cause and treatment are specifically known today, is myxedema. My function this evening will be to apply to neurasthenia, migraine and epilepsy the results of observation and experience in these states from the point of view of endocrinology.

Do you remember the poor victims of Beard's "nerve exhaustion," with their fatigability, their constantly recurring complaints of arrhythmic heart, precordial pain and intestinal stasis; with the seemingly wild subjective descriptions of their constantly changing state; with no friend or physician to understand them or help them, and who, compelled to go their way alone and misunderstood, were finally left at the mercy of the "patent medicine" output of the country? We laughed at their complaints, scolded them, and in the meanwhile grew gray-haired. But we rarely helped them. Our effort was the one constantly repeated assurance—given with pill or tonic—"it is only a matter of a few years, and the thing will pass over leaving you as well or better than before." We deprived them of their coffee, their tea, their tobacco and their alcohol; we changed their surroundings and their habits, but we failed to change their religion. This they did themselves to remedy our neglect and became Christian scientists.

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* Read at the fortieth anniversary of the Boston Society of Psychiatry and Neurology, Jan. 15, 1920.

Do you remember the classic case of migraine that greeted us with our first patient of a busy day? He had been to ever so many physicians, each of whom had some new method for relieving him. Did you not inquire into his family history almost from the first and grasp with avidity the fact that his paternal grandmother had had the same kind of headache? And that the headache was relieved as soon as he had vomited, and that he really couldn't remember that any particular food was a necessary accessory before the fact, and that perhaps for a short while his vision had been disturbed, or his eyelids may have drooped? And a little more of this, and you were at the end of your tether, your questions came more slowly and finally stopped altogether. You maneuvered to find out what others had done for him, so that by no chance you would make a faux pas; then you made a pass in the dark—to no avail—and your scalp was added to those in his belt.

And we certainly have with us, even today, our constant companion, the patient with idiopathic epilepsy—unless, perchance, he has made newer friends among the abdominal surgeons, or the dentists or the psychologists. He was the bane of our calculations. He rarely had his attack when we thought he should; he would surprise us by freedom from attacks and give us credit for it, when we knew better; and bromid was his portion, however disguised, with an occasional treat in the way of crotalin, rattle-snake poison, or solanum carolinense. Shades of a benighted era! How low had we fallen!

But gentlemen, our attack on these enemies has taken another turn. Our tactics have changed. What we have heretofore regarded as entities are found to be merely incidents. And combined with numerous other incidents, though perhaps less manifest and hence requiring some search, a fairly completed disease picture can often be pieced together under each of the foregoing groups.

NEURASTHENIA

Our neurasthenic patient, according to our heretofore accepted examination, showed, neurologically speaking, perhaps a slight change in the reflexes, very little change from the normal in his urinalysis, and perhaps a slight anemia in the usual blood examination. His viscera seemed normal and even his blood pressure was not far wrong. We therefore told him there was no organic lesion, and perhaps reserved to ourselves the thought that his was some defense reaction against his environment. Of a certainty, therefore, there was nothing to cure but his maladjustment. But not one of us is ever properly and entirely happily adjusted, and yet we do not all become neurasthenics. Why? If we had pursued our examination somewhat farther we would have found a number of curious facts, seemingly irrelevant, which repeated themselves in a majority of our cases. These facts I will endeavor to group successively. The first embraces the early history of our patient. He was late in cutting his teeth; he suffered from nose-bleed; he was a bed-wetter to the tenth year. A few years later he began to grow rapidly and began to have headaches. He had muscular cramps and spasms, "growing pains"; his arms looked like flails. His secondary hair—both on face and in the pubic region—came rather late. He was usually affected by cold weather, and his extremities were cold and damp. As his full growth was attained, these signs gradually disappeared and to all appearances he was well and sound. He married, was happy for some years, and then gradually he began to feel that he was somehow losing ground. He had a bronchitis, following which he began to lose vigor and initiative; he felt tired at slight exertion, his appetite began to wane, he lost weight. He knew that somewhere in his economy things were wrong; he felt his pulse, he sensed his heart throbbing in his arteries, he became so occupied with his search that any disturbing factor or even ordinary conversation made him irritable, and from that time on he began his neurasthenic stop-over journey around the medical world.

Now, however, we will give some facts of his present examination that are pertinent and that will explain many of his subjective complaints. Stripped, he will show disproportionate skeletal growth; arms too long for the body, a thorax too short for the legs or vice versa; teeth that are somewhat anomalous in that the lateral incisors are small and possibly decayed, the canines with attributes of incisors; hairy growth may disclose a feminine pubic distribution or other abnormal features. The scrotal fold may surround the base of the penis. The reaction of the skin to stroking will frequently, especially on the abdomen, show a white line, while on the thorax it will be pink. The blood pressure, if taken casually, may be found to be normal or even somewhat higher. But taken as it should be, namely, two or three successive systolic readings at minute intervals, a rapid systolic drop may be seen, the final reading being below normal. I have seen systolic readings taken in this way to be as different as 175: 130: 105: with a diastolic reading of 70. There is a general relaxation of the smooth muscle organs and of the tendons, so that flatfoot, sacro-iliac backache and visceroptosis are present. The laboratory report of the blood will show an almost normal red and white cell count and hemoglobin content. But if a more intensive examination is made, it will be found that there is a decided drop in the polymorphonuclear count and a relative lymphocytosis; the carbon dioxid tension verges to the lowest normal, the sugar content is abnormal, the blood coagulability is delayed, and often there is an eosinophilia. Even with these few salient points in our examination, it will be seen that our attack is quite different from the old method. The interpretation of this picture is a faulty compensation of a suprarenal inefficiency. This suprarenal inefficiency has arisen as a result of some final causative factor, such as an infectious disease, but on a preformed basis of endocrine instability. The life history of the patient gives manifold evidences of such disturbances in growth and metabolism especially. Such suprarenal inefficiency may remain latent until the patient subjects himself to stress and strain beyond his narrowed limits of fatigue, or may be partially compensated for by other glandular structures, such as the pituitary. Overcome this compensation, and we have manifestations of neurasthenia, psychasthenia, and the psychoneuroses. Definite improvement may be obtained by appropriate therapy, based on each patient's individual incapacity for self-compensation. The very agents that heretofore were forbidden our patient—coffee, tea and alcohol—are extremely useful adjuvants in this treatment. The actual glandular therapy cannot be considered this evening.

MIGRAINE

Our attention is next drawn to our migrainous friend. If we dwell on his family history and antecedents, not only do we find attacks of similar nature, but several other points of significance. Many of the ancestors are tall—over 6 feet; some may have had diabetes; occasionally we find a goitrous person, and if the history is thoroughly looked into, many other endocrine anomalies may be disclosed. Again, during the early life of the patient, features of a disturbed endocrine balance are manifested, especially those of a dyspituitarism. Hair anomalies are frequent; disproportion in skeletal growth likewise; gonadal development is usually tardy. Girls menstruate rather late, and their periods usually are delayed. The actual migrainous attack quite frequently hinges about the menstrual period. Considering together some of the outstanding symptoms of a migrainous attack, we are struck by the fact that almost all can be accounted for by a disturbed pituitary body. There is a change in the blood pressure, which is usually quite low during the attack; a change in the blood sugar content; a retarding of the pulse rate, frequently down to 40 a minute; and at first a lowering of the temperature. The beginning of the attack with polyuria, the frilosity, the vasomotor spasms, all indicate pituitary involvement. The yawning which frequently precedes the attack is usually an accompaniment of fatigue on a basis of primary suprarenal and secondary pituitary deficiency. If we examine the skull of our patient with the roentgen-ray, we will almost invariably find evidences of a small sella turcica, with erosions. The production of the migrainous attack is about as follows: At periodic intervals, such as menstrual recurrences, the pituitary body enlarges. Other factors producing such enlargements are fatigue, which, drawing out the available epinephrin supply, calls on the pituitary to compensate for the purpose of maintaining a proper blood sugar supply and a proper blood pressure; or intense cold, which, causing constriction of the superficial vessels, produces an enlargement of the deep lying organs, among them the pituitary; or intense mental occupation, which likewise produces an engorgement of the cerebral vessels, including the highly vascular hypophysis. Ingestion of certain kinds of food, notably carbohydrates in quantity, may also cause enlargement of the gland. If the sella turcica of a person subjected to these disturbing factors is too small to accommodate the enlarging gland, pressure against the surrounding structures of the hypophysis occurs. First, there is erosion of the surrounding bony capsule, and then pressure against the softer structures beyond. Such pressure occasions headache. This may be unilateral or bilateral, but is always at first referred to the intratemporal region. If the enlargement continues, the pressure begins to be exerted on either the cavernous sinus or anteriorly on the optic chiasm. But the cavernous sinus is in intimate relationship with the third, fourth and sixth cranial nerves, the ophthalmic division of the fifth with its lacrimal branch, and the sympathetic plexus of the carotid. Furthermore, it drains the ophthalmic vein. Is it merely a coincidence that migraine, as it progresses, involves practically all these structures, producing oculomotor palsies, disturbed lacrimation, hemianopsias, intra-ocular pain and pressure and perhaps exophthalmos, and finally, pressure on the carotid plexus, and universal headache? But you will say, if this is so, surely such conditions must occasionally be seen at necropsy. They have been seen. Deyl, at a Paris international congress a few years ago, reported several cases of migraine in which the postmortem examination showed a unilateral pressure exerted by the enlarged hypophysis on the cavernous sinus. I, myself, have among my roentgenograms, four that show a very marked unilateral erosion of the clinoids in patients suffering from migraine. About ten years ago, Plavec confirmed Deyl's findings. Hence in migraine, we have symptoms referable in the first place to metabolic disturbances caused by the pituitary, and secondly, to the direct pressure exerted mechanically by the enlarged gland. Whether those cases in which the headache begins in the occiput and which are occasionally accompanied by hemiparesis or hemianesthesia are of this same class, remains to be proved. For the present, it would be well to classify the migraines on some such basis as I have indicated. It seems to me that this conception of the typical migraine attack with ocular symptoms is the rational one and explains practically the entire situation.

EPILEPSY

When we consider epilepsy, our problem changes somewhat. We must first classify our cases. Which are frankly endocrine, and which are not? Our criteria are found in the hereditary history, the developmental years and the present examinations. To give you all the endocrine stigmas to be encountered in these three groups would be to review the subject of endocrinology. Briefly, the grosser hereditary factors of importance are: abnormal skeletal growth, pronounced abnormal quantity and distribution of hair, obesity, diabetes, goiter and fatigue states; vasomotor disturbances, such as urticaria, Raynaud's disease, erythromelalgia and intermittent claudication. Pronounced cases of such complaints in the family must awaken the suspicion of an endocrinopathic heredity. The early life of the patient must show some of the endocrinopathic tissue changes or metabolic disorders for which we searched his heredity. There will be in his adolescence signs of fatigability, drowsiness, headaches; precocious or tardy genital development, invert signs of secondary sex characteristics, such as breasts in the male, mustache and male pubic hair in the female. In our examination of the patient, we must note each tissue, bone, muscle, skin, hair, nails and teeth. Is the jaw prognathous, are the teeth spaced or crowded, is the palate narrow and high or broad and shallow, is there much or little hair and is it properly distributed? Is the ratio of torso to leg normal; are the hands long and slender or broad and chubby? Are the skin reactions normal to stroking? Is the blood picture correct or have we a relative lymphocytosis, or an eosinophilia, or a low blood sugar content? Is the coagulation time between five and eight minutes, as it should be? What is the carbon dioxid tension? Blood pressure, temperature and pulse rate may also be suggestive. Is the thyroid large, or does the roentgen ray show any sella turcica abnormality? If our epileptic patient withstands to a great extent these inroads on his endocrine assets, then is he not of our present class. Those that fall well within our suspicions can be divided into two groups. The first is represented by the adipose, sluggish individual with smooth, velvety skin, body hairless, small genitals almost imbedded in the lower fatty abdominal folds, with a high sugar tolerance and a small closed-in sella turcica. This group represents the type of which Froelich's dystrophy is the classic. There are many variations with a corresponding change in the physical signs. Those of this group that have epileptic seizures—and there are many—frequently have the uncinate fit with aura of smell or taste. They are distinctly pituitaric. Feeding them with appropriate doses of pituitary gland, frequently the anterior lobe extract only, produces marked improvement and even cures the disease. The epileptic fit is produced as a symptom in this state, much as is the migrainous attack mentioned in the foregoing. A periodic enlargement of the hypophysis, produced as before, in a sella not roomy enough to accommodate it, causes the critical moment. At times, in these cases, a migrainous attack accompanies the seizure, or is the equivalent of a seizure, and the uncinate features are presumably due to the extension of the pressure to the uncinate gyri of the temporosphenoidal lobe immediately surrounding the sella. This serves to explain the fact of the interconvertibility of migraine and epilepsy, cited forty years ago by Gray—an observation which in the experience of each one of us has had its examples.

The second group is represented by persons, likewise of endocrinopathic heredity and early life's history in whom the attack is secondary, in all probability, to retained nitrogenous products — presumably split protein and amino-acids. The retention in the tissues, in fact even in the individual cells, of these partial decomposition products, acts as an irritant which finally produces the epileptic seizure as a purposeful act; for the convulsion, accompanied by its profuse sweating, increased kidney and bowel action, tends to relieve this condition. The reason for the retention of amino-acids—partial protein decomposition products—in the cell, has been described ably by E. C. Kendall of the Mayo Foundation. He has shown that the iodothyroid product of the thyroid gland is instrumental in converting the amino-acids into ammonium carbonate, carbon dioxid and water, the ammonium carbonate being probably changed by parathyroid activity into urea. In these final forms, the protein combustion products are ready for excretion by the emunctories. Failure of the thyroid-parathyroid apparatus, then, means failure in the removal of the toxins. This situation determines the seizure. It is our task, therefore, to determine in which of our epileptic patients this condition obtains. I do not believe that there is a constant subthyroid state that is the goal of our quest. Such a state would determine a generalized hypothyroidism, a partial myxedema. A myxedematous state more or less is self-compensatory in that the person is sluggish, slow of mentality and activity, with a much lower basic metabolism than is normal. He burns less tissue, and there is less to excrete. But our dysthyroid epileptic is one in whom either periodically or after a period of excessive oxidation, the thyroid-parathyroid apparatus is insufficient. In other words, an instability of this glandular activity must be predicated. Such instability, however, is almost never primary, depending on its covariants among the other endocrine organs for its production. A marked ovarian disturbance, a suprarenal outpouring, an underfunctioning pituitary — all may serve to make excessive demands on this thyroid-parathyroid organ, allowing insufficient secretion for the performance of its catalytic activity. This constellation of the units of the internal glandular system determines the attack. It is not a condition of pure hypothyroidism as it is pictured by some authors. Hence the stigmas of hypothyroid states are not successively found in these subjects. One may just as frequently find ovarian or suprarenal or pituitary characteristics. The main points to determine are the endocrinopathic family history and the presence of glandular disturbances in the early life of our patient. The treatment also would vary considerably depending on the result of our investigation into the primary disturbing element. If this could be found, and corrected, the seizures would cease. Frequently this can be done. Ovarian or gonadal disturbances are often amenable to treatment. Emotional states, fright, fatigue, anger, serve to call out epinephrin in too great amounts not to involve overactivity of the thyroid; but these conditions are difficult to combat. It is here that thyroid administration is indicated. Such administration should neither be continuous nor given in large dosage. Large doses would simply serve to increase the protein disintegration and would tend to increase the seizures. It is in these cases that proper administration reaches its highest art.

COMMENTS

I hope I have indicated in this comparatively short sketch, in which only the high lights could be pointed out, the bearing of endocrinology on some of our old problems of neurologic interest. You see, new points of attack are disclosed, and our patients are viewed, not as representing entities in their disease complexes, but as being simply the results of various combinations and permutations of constantly varying factors; their symptomatology, while manifestly widely differently constituted, has many basic similar ground plans. They must always be treated individually and as individuals, and never as group cases with definite limitations. Endocrinology, gentlemen, marks a new era in medicine.

MONOPLEGIA SPINALIS SPASTICA *

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We have had in the neurologic service of the University of Pennsylvania a number of patients in whom paralysis of one lower limb was associated with atrophy, exaggerated tendon reflexes of the affected limb and in some instances with spasticity. The first case of this kind was reported by Dr. Williams B. Cadwalader,[1] June, 1916, and is given as Case 2 of this paper. Since that time a considerable number of cases have been under observation, but it was not possible to devote sufficient time to scientific work during or immediately following the recent war. This form of monoplegia has also been studied by Sittig[2] and in all his cases the upper limb was affected. He had no necropsies. He suggests the term monoplegia spinalis spastica superior. In all my cases the lower limb has been the one affected, so that a lower as well as an upper type occurs, a monoplegia spinalis spastica inferior. It seems preferable therefore to describe the condition simply as monoplegia spinalis spastica, or in the English equivalent, spastic spinal monoplegia.

In some of Sittig's cases sensory disturbances were observed on the ulnar side of the paretic upper limb from a bullet wound of the cervical region. He found that the syndrome may result from different forms of spinal paralysis, from diplegia, hemiplegia or the Brown-Séquard complex, but it may be primary from the beginning. The condition differs from that in which there is hemiplegia from a spinal cord lesion in which the paralysis of the upper limb is merely more pronounced than that of the lower. He believed the explanation awaits further investigation. Fabritius came to the conclusion that the fibers for the upper limb are separate from those for the lower limb in the crossed pyramidal tract of the spinal cord, but Hoche, Fischer and Gierlich have shown by microscopic study that small lesions of the anterior central convolution of the cerebrum cause diffuse degeneration of the crossed pyramidal tract of the cord by the Marchi stain.

The most striking case reported by Sittig is one in which spastic monoplegia of the left upper limb existed two years after a bullet wound of the neck had been received, while the other limbs had full

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* Read before the Philadelphia Neurological Society, Jan. 23, 1920.

[1] Cadwalader, W. B.: J. Nerv. & Ment. Dis. **43**:547 (June) 1916.

[2] Sittig: Monatschr. Psychiat. u. Neurol. **46**:112 (Aug.) 1919.

movement and sensation was not affected. Sittig had no pathologic findings to offer in explanation, but he believed there must have been a partial transverse lesion, probably an area of necrosis, in the upper cervical cord, and that it could not have been a lesion of the anterior horn, because of exaggeration of reflexes in both upper and lower limbs, especially in the upper, and because of absence of pronounced reaction of degeneration. The lesion could not be cerebral, as indicated by the course of the bullet and by the fact that in certain cases the symptoms must be of spinal origin. In one case the interossei muscles of the hand were atrophied and there was slight change of electrical reaction. There was not spasticity in all the cases Sittig reports. In Case 3 he distinctly says the tonus of the upper and lower limbs was normal; in some other cases of his series it was only a little exaggerated. In Case 7 both upper limbs were weak and the tonus of these limbs was normal. In Case 8 the tonus of the weak right upper limb was not increased.

There is some danger that spastic monoplegia of cortical origin from a lesion of the cerebrum may be mistaken for one of spinal origin, and this possibility should be kept in mind. The cerebral form has recently been the subject of a paper which I contributed to the anniversary volumes dedicated to Sir William Osler.[3]

Some of the cases of monoplegia with exaggeration of tendon reflexes result from injury of joint or bone in the affected limb, or from a condition which Babinski has called reflex paralysis. Where the bone or joint is affected there is likely to be pain, and spasticity is not conspicuous. Such a case is number 5 in my series, and it is reported because of its clinical resemblance to the other cases.

Even where the lesion is one of the spinal cord the spasticity may be slight, as in most of my cases, or pronounced as in my Case 7. The explanation which appeals to me for the spastic spinal monoplegia is one I [4] advanced in 1907. At a meeting of the Philadelphia Neurological Society on November 26 of that year I reported two clinical cases of poliomyelitis with exaggerated reflexes, gave references to a few similar cases in literature, and suggested an explanation for atrophy with spasticity and exaggerated reflexes of spinal origin on the supposition of a lesion resembling that of amyotrophic lateral sclerosis, i. e., with degeneration of both pyramidal tract and anterior horn of the spinal cord.

I described a case I had published in *Brain* in 1903, one of acute poliomyelitis with necropsy. The degeneration of the lumbar region was typical of this disease and was confined to the anterior horns. Implication of the lateral columns occurred in a limited area in the thoracic region. The case offered an explanation for the exaggeration of tendon reflexes in poliomyelitis. It seemed to me that if the degeneration of the lower part of the cord does not implicate the cells innervating the thigh muscles, but is confined to those supplying the leg muscles below the knee, as is so often the case; and if the lateral column of one or of each side is implicated in the thoracic region, exaggeration of the patellar tendon reflex may occur. This point of view has received corroboration by a case of poliomyelitis with necropsy I have recently studied.

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[3] Spiller: Contributions to Medical and Biological Research Dedicated to Sir William Osler, J. Nerv. & Ment. Dis. **35**:258, 1908.

[4] Spiller: J. Nerv. & Ment. Dis. **35**:261, 1908.

REPORT OF CASES

Case 1.—*History.*—J. W., aged 62, in the service of Dr. C. K. Mills at the Philadelphia General Hospital, was admitted Dec. 31, 1917, and died Sept. 22, 1919. The right lower limb had been wasted since he was 2 years old. The limb was not only smaller but it was also shorter than the left. The statement was made that when he was 2 years of age he was taken outdoors by his sister, and when he was put down to walk his right lower limb gave way. Since that time the condition of this limb had been abnormal.

Examination.—The irides contracted sluggishly to light but promptly in accommodation. He had had a + + + Wassermann reaction. The grip of each hand was fairly strong. The right lower limb was slightly hypertonic. The triceps, biceps, patellar and Achilles' reflexes were all exaggerated on each side. No sensory changes were observed. The man was feeble in his limbs.

Necropsy Examination.—The lumbar and sacral roots could not be counted on account of the condition resulting from the necropsy. The lumbar and sacral regions were divided into blocks about 3/16 of an inch in thickness and sections were taken from each block. The upper part of the second lumbar segment appeared to be normal, but in the block immediately below this the greater part of the left anterior horn was degenerated and nerve cells and medullated nerve fibers had disappeared. The right anterior horn at this level was normal. The Marchi sections showed no distinct recent degeneration.

In sections from the next block the left anterior horn was normal, so that the degeneration of this side was limited. The right anterior horn showed a normal appearance as regards medullated nerve fibers but contained few nerve cells. From this level downward the degeneration of the right anterior horn in its outer portion was pronounced. In the upper levels the degeneration was confined to the outer portion, but a little lower the outer half of the right anterior horn was degenerated. In its inner half medullated nerve fibers could be seen, evidently coming from the left anterior horn through the anterior commissure. The degeneration of the right anterior horn extended from the middle of the lumbar region through the sacral region to its middle portion. Both crossed pyramidal tracts appeared slightly degenerated. A moderate degree of mononuclear cellular infiltration was found in the pia of the spinal cord and over the medulla oblongata, and this in connection with the Wassermann reaction and the sluggish iridic reflex to light suggested that the slight degeneration of the lateral columns probably was caused by syphilis. In addition, a small old area of softening was found in the right motor tract of the pons.

The exaggeration of the tendon reflexes probably was caused by the degeneration of the lateral columns, but as many of the cells of the right side of the cord in the outer half of the anterior horn of the lumbar region were destroyed and there were numerous medullated fibers in the inner portion of the right anterior horn in the lumbar region which came from the left anterior horn, it would appear that the exaggeration of the tendon reflexes in the right lower limb was partly dependent on these nerve fibers from the left anterior horn.

Case 2.—This case was presented by Dr. Williams B. Cadwalader in 1916 with the title "A Case Presenting Atrophy of the Right Lower Extremity with Increased Reflexes and Positive Babinski Sign."

History.—V. J. S., a boy, aged 17, was admitted to the University Hospital on Jan. 13, 1916, complaining of weakness and wasting of the right lower extremity. The patient's father was said to have had syphilis; his mother, two older brothers and two younger sisters were living and well. The patient stated that he had been well until 1913, when he had had an attack of what he had been told was rheumatism. This condition was characterized by pain on movement in the joints of both lower extremities and also in the right hand. There was no redness or swelling. After a few weeks he made a complete recovery. In September, 1914, he again had some pain in the region of his hip joints which was increased by movement. It disappeared, however, after a few days. He stated that since September, 1914, his right lower limb had been gradually growing weak and the muscles had been wasting. In all other respects he was entirely well.

Examination.—His eyes, cranial nerves, upper extremities and internal organs were found to be normal. The muscles of the right lower limb were moderately and uniformly atrophied and there was weakness in proportion to the wasting. The tendon reflexes were exaggerated, more so on the right side than on the left. There was a distinct Babinski sign on the right but not on the left side. Ankle clonus was absent. Sensation was normal. There was no incoordination, ataxia or tremor.

Examination of the joints by Dr. Edward Martin revealed nothing abnormal and in his opinion the atrophy was not related to joint disease. This was later confirmed by Dr. Pancoast who made roentgen-ray studies. Dr. Leopold made an electrical examination and reported that the muscles reacted normally to the faradic current. The Wassermann reaction was negative. Blood count and urine examination were also negative.

The combination of atrophy of gradual onset with increased reflexes and Babinski sign Dr. Cadwalader believed was to be explained by an infection which had caused pain in the joints simulating rheumatism, and had implicated the spinal cord as in acute anterior poliomyelitis. Some of the anterior horn cells probably had been destroyed and the pyramidal tracts had been involved.

Case 3.—*History.*—H. B., aged 8, was brought to the hospital by his mother, Feb. 12, 1919, because she had noticed that the left thigh was becoming smaller than its fellow. This was first noticed two months before. One year earlier the boy was run over by an automobile and his left leg was injured; he was obliged to remain in bed six weeks at that time. He said that he had not had any pain in the left hip or thigh, and his mother said he had not had any febrile condition during the past year. He had one brother and one sister, both living and well.

Examination.—The pupils, the ocular and other cranial nerves were normal. He had no signs of progressive muscular dystrophy. The entire thigh and leg on the left side were smaller than the right lower limb, and the thigh was more affected than the leg. The patellar and Achilles' reflexes were present and exaggerated; the patellar reflex on the affected side was more exaggerated than the reflex on the other side. He had no Babinski sign and no ankle clonus. There was no change of sensation and no evidence of hip or vertebral disease.

Diagnosis in this case was difficult. The atrophy and exaggerated patellar reflex of one limb might permit the diagnosis of arthritic muscular atrophy, but as there had been no pain and there was no sign of joint or bone disease the possibility of a lesion of the spinal cord could not be excluded.

Case 4.—*History.*—T. B., aged 19, was admitted April 25, 1919. There was no history of a similar disease in the family. He had always been well except for measles and mumps in childhood. He had no history of injury or disease before the atrophy was observed. He came to the hospital because of weakness and atrophy in the left lower limb. He noticed the weakness first in February, 1918, when he was doing endurance tests in the army, as he was a soldier. Soon after these tests he noticed that the left calf was smaller than the right.

Examination.—The circumference of the left calf was 2 inches less than that of the right. He flexed the left foot on the leg poorly, but had no difficulty in movements of the right foot. The left leg was markedly weak in resisting passive movement at the knee. The left patellar reflex was exaggerated but the left Achilles' reflex was not elicited. He had no clonus nor Babinski sign. The right tendon reflexes were normal.

While in France he carried about 70 pounds and did much hard marching over obstacles. At these times he had pain in the region of the left hip. The left sacro-iliac joint was photographed and was normal. The left foot was much smaller than the right, as if the bones had not developed.

The smaller size of the left foot suggested arrested development in childhood from poliomyelitis, especially as the left Achilles' reflex was absent. The severe strain caused by the endurance tests of the army may have caused the atrophy of the lower limb already inadequately innervated on account of the poliomyelitis. It is well recognized that atrophy may develop from old poliomyelitis, especially when the affected limb is put to excessive use. The exaggeration of the patellar reflex suggested further spinal disorder. There was no reason for a diagnosis of arthritic muscular atrophy, and yet there are certain superficial resemblances between this case and the following one in which the diagnosis of arthritic muscular atrophy was correct.

Case 5.—*History.*—H. L., aged 25, was referred to me by Dr. Adlai S. Oliver of Benson, N. C., April 4, 1919. He had seen the patient only the day before he referred him.

The man was a soldier in France in the Engineering Corps when the symptoms began. He was obliged to take walks from 15 to 25 miles carrying from 70 to 80 pounds, and while doing this his right lower limb would give out. Symptoms began with pain in the right ankle in January, 1918, and not until six months later did he notice wasting and weakness of the limb. After about a month the pain seemed to be referred to the right hip joint.

Examination.—Examination at this time showed that the right calf was about ½ inch smaller than the left. A diagnosis of tumor of the spinal cord was made. Roentgen-ray plates of the spinal column and hip were negative. The Wassermann test, presumably of the blood, was repeatedly negative. He had no history of disease, either acute febrile or venereal.

The right lower limb was wasted. The thigh measured 2 inches less than the left in circumference, and the right leg 1 inch less. The whole right lower extremity was somewhat weak, but the man still had good use of the limb. The tendon reflexes were equally exaggerated in the lower limbs, but there was no ankle clonus. There was no Babinski sign and no disturbance of tactile, heat, cold, pain or deep pressure sensation in the lower limbs. There was no spasticity. He had become progressively worse.

An area of intense tenderness to slight pressure was found at the lower posterior and outer part of the right tibia. When examined by me the patellar reflexes were much exaggerated and equally so; the right Achilles' reflex was exaggerated, the left was about normal. The pain was constant in the right foot and usually increased by walking.

I strongly suspected some periosteal disease and sent him to Dr. Pancoast for roentgen-ray examination. He reported that there was an area of chronic localized osteomyelitis (infective type) at the lower end of the tibia. The lesion might easily be overlooked. He recommended operation.

Case 6.—*History.*—F. C., aged 34, a carpenter, came to the hospital Jan. 26, 1920. His wife had had two children and no miscarriages. He had gonorrhea when 24 years old and had used alcohol moderately. Five years before he began to have pain in the right lower limb which at first was like a sensation as of pins and needles, and was felt chiefly in the anterior aspect and in the posterior part of the knee. He had no weakness but could not walk very well at that time because of the pain. He described the pain as a burning like fire, and it was worse at night. This pain lasted about a year. The right calf was operated on, and he had a large scar. The wound did not heal readily, and a year was necessary for recovery.

He had no more symptoms until ten days before he came to the hospital. He had been working as usual and one night when he came home from work he found he could not use the right lower limb in walking, and this was so weak he could not lift it. He had no pain and has not had any. He had no disturbance of the bladder.

Examination.—The right pupil was a little larger than the left but reaction was prompt. The face and upper limbs presented nothing abnormal. When he walked he had great difficulty in supporting himself on the right lower limb which he dragged along. It was smaller than the left, both in the thigh and calf muscles. He had no power in flexing the right foot on the leg, but had slight downward movement of the foot and was able to move the toes a little. He had little power in the right hip and knee, and when he uncrossed the right lower limb from the left knee the right limb fell to the floor. The patellar and Achilles' reflexes were exaggerated, and those on the right side were more exaggerated than those on the left side. Ankle clonus was persistent on the right side and abortive on the left side. Babinski reflex was typical on the right side and almost as typical on the left. He moved the left lower limb freely and apparently without loss of power. Tactile sensation was normal everywhere, but the point of a pin was miscalled at times in the left lower limb, as was also heat and cold stimulation in this limb. Vibratory sense and sense of position were normal.

Course of Disease.—The man returned Feb. 2, 1920. His condition had changed greatly. He had marked increase of power in his right lower limb and could move the limb more freely. He had no disturbance of sensation. Ankle clonus and the Babinski sign were still present on the right side. The right thigh measured 38 cm., the left 41 cm.; the right leg measured 31 cm., the left 32 cm. The right lower limb was slightly rigid. The Wassermann test was reported delayed negative, less than ¼ inhibition.

On February 9 he had improved still more and had a great deal more power in his right lower limb, although he said both lower limbs seemed a little weak. There was now no decided loss of power in either lower limb.

The case evidently was one of organic change in the right side of the spinal cord, as shown by the ankle clonus and Babinski reflex. He probably had syphilis and syphilitic myelitis, and as a result of overwork the diseased blood vessels of the right side of the cord became congested. When he stopped working he allowed the circulation in the spinal cord to be restored in a considerable degree, but he was still weak in both lower limbs. The right crossed pyramidal tract and the right anterior horn of the lumbar region probably were affected.

Case 7.—*History.*—S. M., aged 33, came to the hospital Feb. 11, 1920. He had paralysis of the right lower limb which came on gradually two years before and which steadily increased. He had not had any pain. The left lower limb showed no loss of power. He sometimes had difficulty in voiding urine, and for the past year had had sexual impotence. He did not have headache, but he thought his eyesight was failing. He had three children, and his wife had had no miscarriages. So far as he knew, his symptoms had not changed during the past year.

Examination.—He had no Romberg sign. His gait was extremely spastic in the right lower limb, and he walked like a hemiplegic patient, swinging the limb from the hip. The pupils were equal and the reaction was good to light and in convergence. There were no symptoms in his face and upper limbs. The right upper limb was as normal as the left. The right lower limb was very weak and spastic, and when the limb was allowed to fall to the floor from the knee of the examiner it fell with the leg extended on the thigh and as a dead member, whereas the left lower limb fell in a normal manner. The left lower limb may have been a little weak, but not much so if at all. He used the left lower limb in a normal manner. The patellar and Achilles' reflexes were nearly equally exaggerated, but ankle clonus was present on the right and not on the left side. There was no disturbance of touch, pain, heat and cold sensations anywhere. Some of the Wassermann tests of the blood had been positive, and he had had twenty-four intravenous injections, but without improvement.

The right leg 8 inches below the top of the patella measured 13½ inches, and the left 14½ inches. The right thigh measured 3 inches above the top of the patella, was 15¾ inches, the left 17 inches; the right thigh 7 inches above the top of the patella measured 19 inches, and the left 19 inches.

This case seems to be similar to case 6 except that the monoplegia of the right lower limb was of long duration. The paralysis and spasticity of the right lower limb were intense. Babinski's sign was typical on each side. Persistent patellar clonus and persistent ankle clonus were present on the right side but not on the left side; the left patellar reflex was somewhat exaggerated. The left Achilles' reflex was about normal.

The pathologic condition was probably a syphilitic lesion of the spinal cord and did not appear to be of cerebral origin.

HISTOPATHOLOGY OF BRAIN ABSCESS

WITH REMARKS ON INTRASPINAL THERAPY *

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The pathologic changes in brain abscess are, as a rule, so manifest, even on macroscopic examination, that a correct diagnosis offers practically no difficulty, however insignificant the abscess may be. Probably this is the main reason for the scarcity of literature on microscopic brain changes resulting from abscess. Yet studies of the histologic changes are of great scientific interest, for they may clear up some as yet unsettled questions which are not only of academic, but also of practical, value.

Brain abscess perhaps brings to mind a circumscribed lesion more or less walled off by a membrane. Since time is required for a membrane to form, the walling off of an abscess necessarily must be a more or less gradual, subacute or chronic process that ultimately leads to transformation of a portion of brain tissue into a cavity filled with pus. This gradual replacing of the brain tissue by a walled off, encapsulated cavity is lacking in acute suppurative brain lesions. In these the suppurative process is rather diffuse, producing pronounced and extensive changes in the ganglion cells, glia tissue, nerve fibers and even in the vessels themselves.

MICROSCOPIC STUDY OF LESIONS OF BRAIN ABSCESS

Photomicrograph 1 (Fig. 1) gives some idea of widespread destruction. It represents a section of the brain cortex from a patient with a septic purulent meningitis secondary to an acute otitic abscess which ruptured into the brain, causing the patient's death on the twelfth day. The structure of the brain is totally changed. Instead of the regular layers of ganglion cells, there is a mass of strange elements which under low power can hardly be defined. Only blood vessels with somewhat thickened infiltrated walls and narrowed, thrombosed lumen, scattered fibrin fibers, erythrocytes and pus cells can be made out. Under a high power, there can be discerned fragments of somewhat changed nerve fibers, glia cells in a condition of ameboid degeneration and scattered hemorrhagic foci. Areas not directly affected by the pus invasion are much better preserved, though microscopically quite changed. In

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* From the pathological laboratories of Cook County and Psychopathic Hospitals, Chicago.

these areas the blood-vessels exhibit dilated perivascular spaces infiltrated with lymphocytes and plasma cells, while the ganglion cells are either in a state of mild chromatolysis or so-called cloudy swelling, or altogether normal. In short, such spaces exhibit parenchymatous and interstitial changes typical of acute nonsuppurative encephalitis. These are the reactions of brain tissue in diffuse suppuration which has not had sufficient time to become encapsulated and which leads to total destruction of some elements (ganglion cells), partial destruction of others (nerve fibers, glia cells) and progressive vascular changes (hyperemia, thickening and infiltration of the vascular walls, new formation of capillaries, etc.).

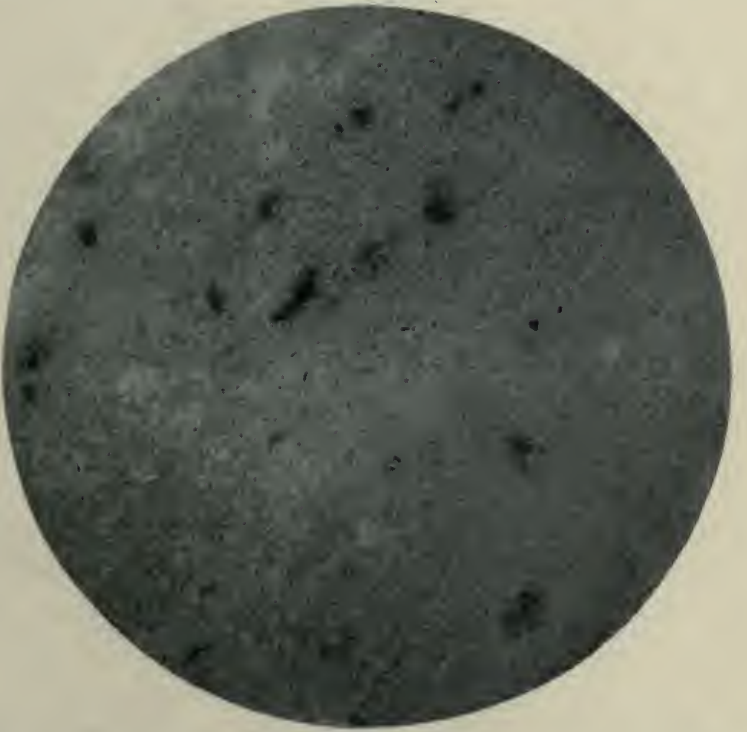

Fig. 1.—Brain section from a case of acute otitic abscess that ruptured into the brain killing the patient on the twelfth day of his illness. The black spots are vessels, thrombosed and with thickened walls, surrounded in some places by fibrin fibers. The numerous dots are glia nuclei. (Frozen section. Alzheimer-Mann stain, × 75.)

Source of Connective Tissue in Brain Tissue.—Perhaps of greater interest are the findings in cases of encapsulated abscess. The capsule of an old abscess may be incredibly thick. For instance, in one of Bassoe's cases of eight years' duration, demonstrated before the Chicago Neurological Society, the thickness was one quarter of an inch, very dense and fibrous, much resembling the dura. In fact, the first impression gained from a superficial examination was that the membrane was an outgrowth from the dura. Thus we are confronted by a peculiar phenomenon: growth of a large mass of hard connective tissue in brain tissue, where under normal conditions, connective tissue is solely confined to vessels and the tender prolongations of the pia. Naturally, it is desirable to determine how and from where such an abnormal, unusual and excessive amount of connective tissue growth develops. This problem I have tried to solve.[1] The study of my material (young membranes) apparently demonstrated that the growth of connective tissue arose exclusively from the blood elements, the lymphocytes, which by gradual and progressive transformation turned

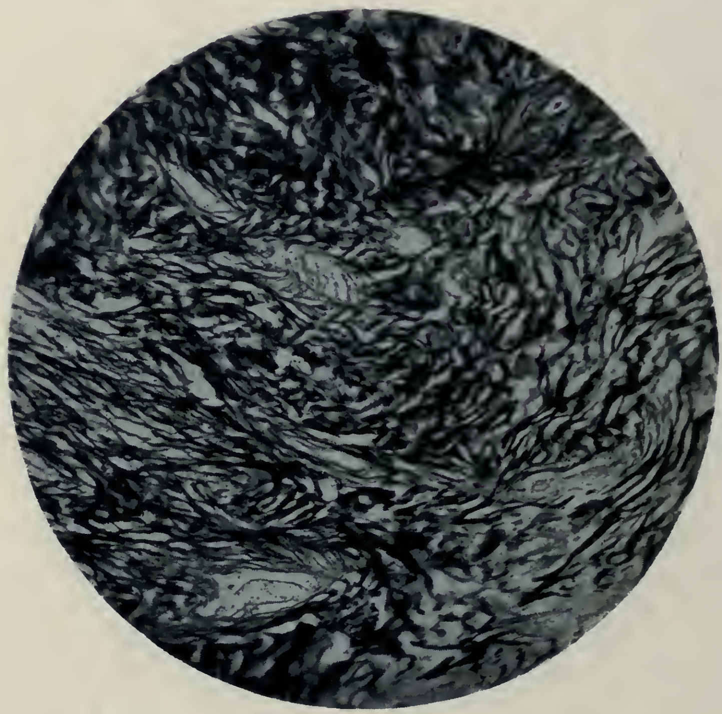

Fig. 2.—A surface (longitudinal) section of the inner layer of the capsule, eight years old. The connective tissue fibers crossing and intercrossing each other form dense meshes with narrow interspaces enclosing a few cellular elements; there is a total lack of blood vessels. The entire structure very much resembles a fibrous scar tissue. (Combined Bielschowsky-Alzheimer-Mann stain. Paraffin section, × 45.)

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[1] Hassin, G. B.: Histopathologic Studies on Brain Abscesses, Med. Rec. **93**:91 (Jan. 19) 1918.

into fibroblasts, and then into connective tissue fibers, finally to become a solid membrane.

Through the kindness of Dr. Peter Bassoe, I have had the opportunity of studying much older abscess membranes. One, about eight years old, enveloped a large abscess that resulted from a bullet lodged in the brain. In three other cases the membranes were comparatively younger, but their macroscopic and microscopic aspects were much alike. The membranes, as well as various portions of the brain and cerebellum, have been studied on celloidin, paraffin, and frozen sections with various staining methods. The capsules were examined in transverse and longitudinal or surface sections, the respective layers having been taken up separately. The inner layer (Fig. 2) directly surrounding the abscess presents a mass of collagen, powerful fibers crossing and intercrossing, forming an irregular network containing

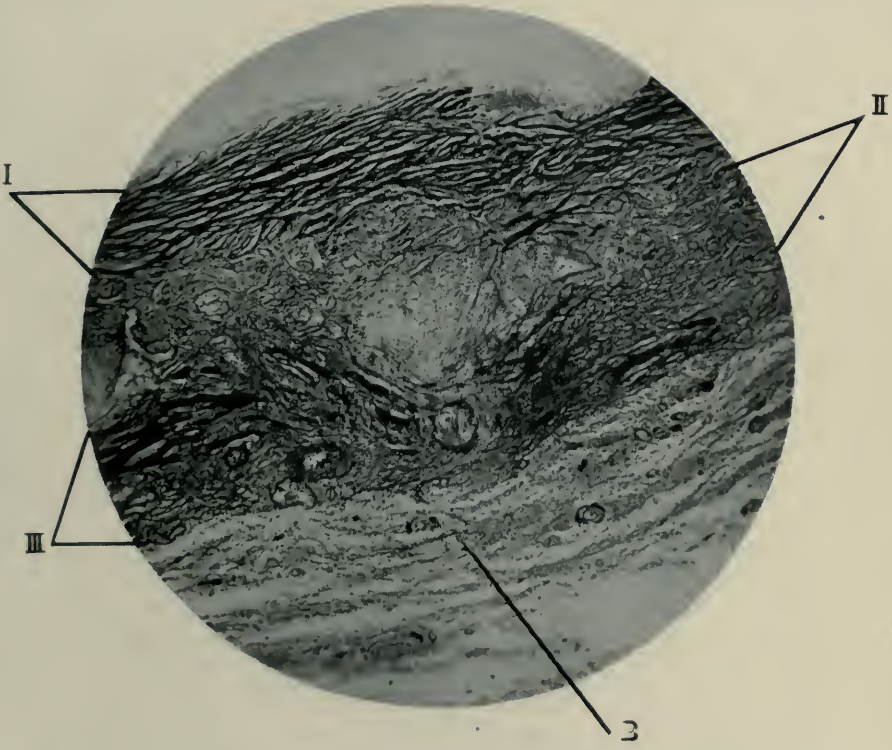

Fig. 3.—Transverse section of the eight year old capsule. The external layer (III) consists, like the inner one, of connective tissue but shows a number of vessels that are absent in the internal layer (I). II—the middle layer also reproduced on a surface section on the photomicrograph. IV—B, brain tissue shows numerous small vessels infiltrated with fat. (Stain same as in Figure 2, × 45.)

numerous interspaces. The latter usually enclose a few cellular elements, or rather remnants of various cells which cannot be well defined. They mostly appear shrunken, some being cystic and vacuolated, some oblong and flattened, with a very pale, hardly visible nucleus. Few of them show the presence of fat. Blood vessels in this layer are lacking, as well as polymorphonuclear cells, pus cells, fibroblasts or other inflammatory elements. Somewhat similar in structure is the external layer (Fig. 3). It also contains a wealth of collagen fibers, which, though somewhat less thick, equally form dense meshes packed with an enormous amount of cellular elements (plasma cells, gitter cells, fibroblasts and some lymphocytes). Vessels here are more numerous, their adventitial spaces are dilated, but blood cells, pus elements, polymorphonuclear cells and signs of inflammation are totally lacking. Between these two membranes there can be discerned (Figs. 3 and 4) the third layer which shows a wealth of cellular elements and vessels. This membrane also contains connective tissue fibers, but these are much more tender and delicate than those of the external and internal layers.

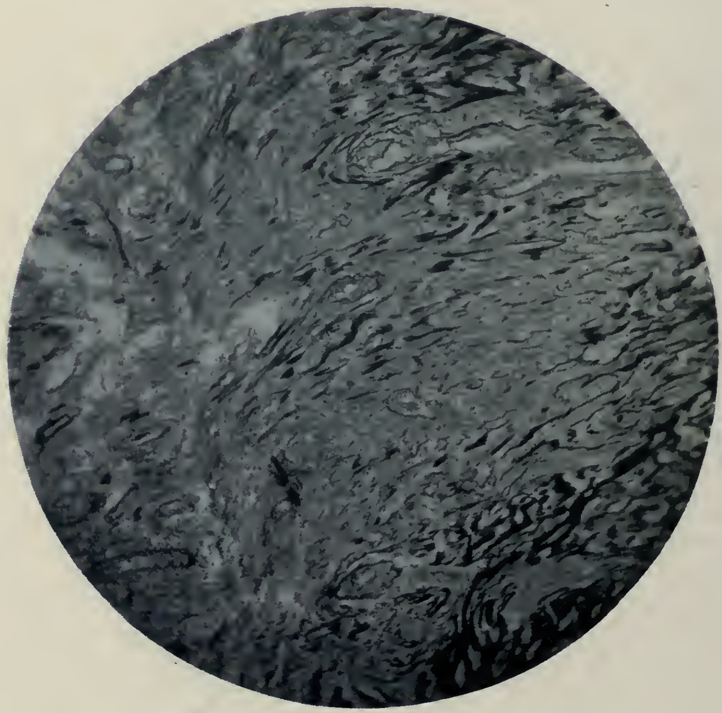

Fig. 4.—Surface section of the middle layer of the eight year old capsule. The predominance of vessels and cellular elements (in the pale areas) is quite marked. (Stain and magnification the same as in Figures 2 and 3.)

In some places it consists solely of a great mass of fat granule bodies which, with Herxheimer scarlet red stain, appear (Fig. 5) as black masses. Being comparatively poorer in collagen fibers and richer in blood vessels and cellular elements (plasma cells, fibroblasts), this membrane is evidently less organized, and therefore less powerful than the outer and inner layers. The difference of structure in the three layers making up the abscess capsule can be seen from a glance at Figure 3 which represents a transverse section of the capsule. The inner and outer layers, similar in structure, are separated by a paler zone rich in gitter cells and blood vessels. The first two appear like a scar, while in the middle the scar formation is hardly advanced. The cicatrization apparently is obtained in those zones where it is most needed: first, in the surface next to the abscess to hold it in check and, second, on the border of the brain substance to prevent its contamination, or invasion by pus. These three layers in a capsule can be still more easily distinguished in a case of but a few months' duration. The membrane in this case (Fig. 6) also shows a powerful inner layer,

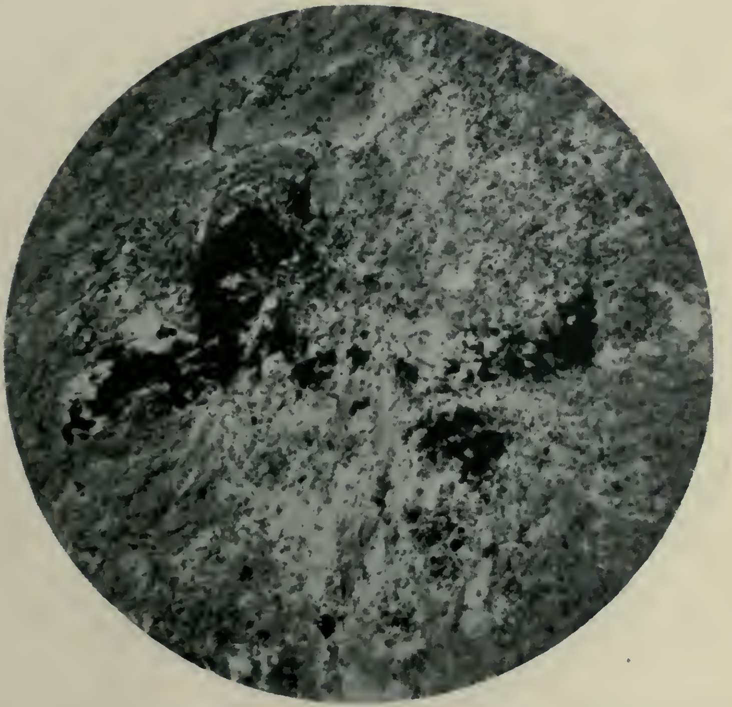

Fig. 5.—Surface frozen section of the middle layer of Figure 3 shows vast accumulations of fat as black masses. (Herxheimer scarlet red stain, × 100.)

a less powerful, though fully developed external layer and a middle layer rich in vessels and cellular elements.

Comparing Figures 3 and 6 one can easily see the difference between these two capsules: the former (Fig. 3) is almost totally organized, while the latter is but partially so. Had the patient in the latter case lived longer, the capsule would undoubtedly have assumed the features of the one in the former case. Whether young or old, once organized, the capsule surrounding the abscess forms an excellent protection for the brain tissue, which is wholly preserved (Figs. 6, 7 and 8). It exhibits no pathologic changes in the ganglions, glia cells or nerve

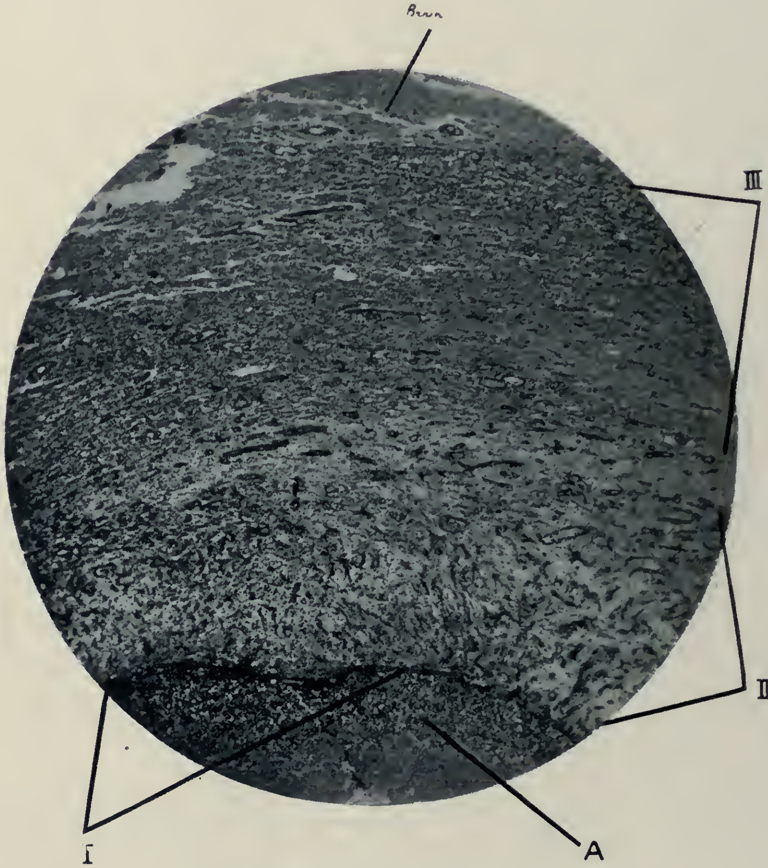

Fig. 6.—Transverse section of the younger capsule (several months old) I, II, III, respective layers; A, the abscess. The top of the picture represents a practically normal brain tissue with several infiltrated vessels. Like the much older capsule reproduced in Figure 2, it shows the identical organization of the inner (I) and external (III) layers; while the middle (II) layer, rich in vessels, is less organized. (Van Giesen stain, × 30.)

fibers, but the vessels and the pia-arachnoid are decidedly altered. While the lumen of a vessel, its intima and the middle layers are normal, the adventitia with its space (Virchow-Robin) is enormously developed (Fig. 9) and made up of numerous bands of connective tissue fibers with narrow interspaces packed with plasma cells. The latter can be easily recognized in the photomicrograph (Fig. 9), especially with the help of a hand lens. Aside from plasma cells and their modifications, young fibroblasts, there are also present gitter cells filled with fat globules. Numerous in the external portions of the adventitia, the cellular elements are scarce in the inner portions, where they are replaced by collagenous fibers, which totally fill up the distended adventitial spaces (Fig. 9). In short, the adventitia is in a hyperplastic state

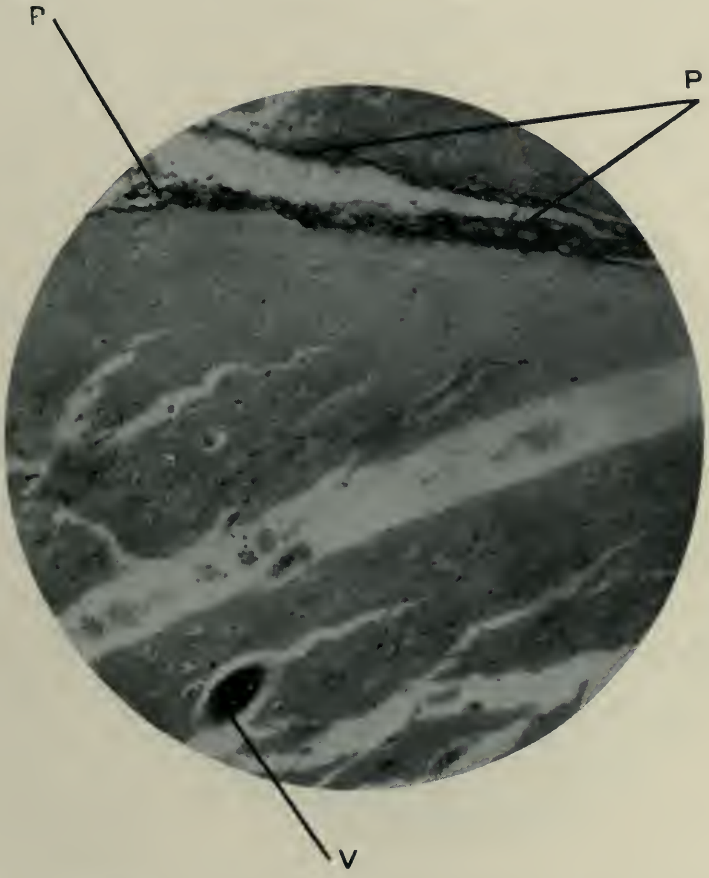

Fig. 7.—Between the much infiltrated blood vessel (V) and the pia (P) (especially its lower branch) is situated an apparently normal brain tissue. The infiltration elements are mostly fat granule bodies and other elements reproduced in Figures 8 and 10. (Bielchowsky-Alzheimer-Mann stain, × 100.)

surrounding the vessels like a muff and invariably containing fat. It represents an extinct inflammatory condition which ultimately resulted in the formation of a fibrous ring around the vessel.

Far removed from such a vessel (Figs. 7 and 8), and separated from it by normal brain tissue, is the pia arachnoid. At the first glance this appears to be infiltrated in some places. However, closer examination (Fig. 8) shows that the infiltration did not involve the pia at all, this membrane being normal. The infiltration is rather confined to the distended subarachnoid space as well as to the numerous vessels. The infiltration elements are partly lymphocytes, but mostly macrophages and gitter cells (Fig. 10). The macrophages and gitter

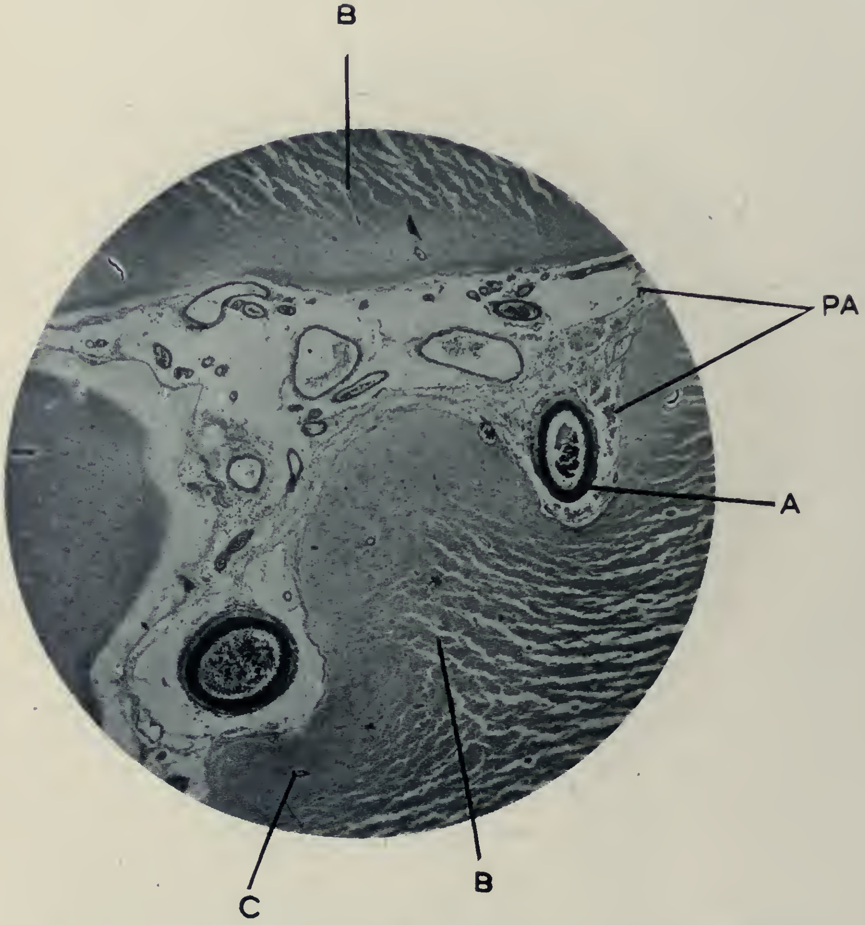

Fig. 8.—P A, pia arachnoid, showing the dilated and infiltrated subarachnoid space as well as numerous vessels; A, artery with an infiltrated adventitial space; B, brain tissue without any changes, except minute capillaries slightly infiltrated with fat. C, infiltrated capillary. Use hand lens. (Same stain as in Figure 7, × 33.)

cells are very large, the former enclosing various débris of lymphocytes, nuclei, etc., while the gitter cells are packed with lipoid, or fat-like substances. Evident signs of inflammation, hemorrhages, fibroblasts, plasma cells, etc., are lacking in the pia-arachnoid. The lipoid substances, as well as cell remnants, enclosed within the macrophages thus can be located in the subarachnoid spaces even when they are widely separated from the abscess and its capsule. The probability is strong that these lipoid substances came into the subarachnoid space from the capsule, its vessels and their perivascular spaces. In other words, it is most probable that the fat found in such abundance in the capsule reached the subarachnoid space by way of the perivascular spaces or—which is the same thing—that the contents of the perivascular spaces were emptied and discharged into the subarachnoid cerebral space. In no other way could the presence in the latter of gitter cells, macrophages and lymphocytes be explained, as a normal pia-arachnoid is totally devoid of such elements.

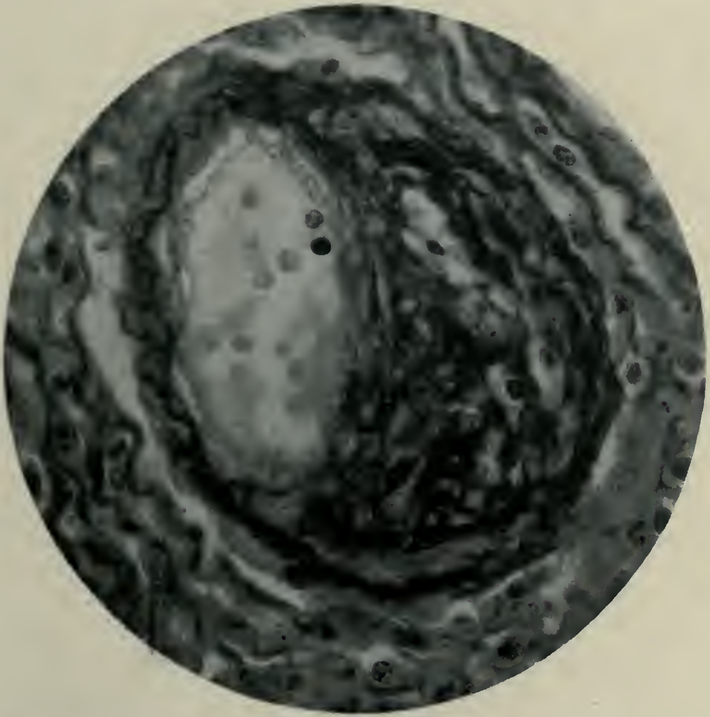

Fig. 9.—Transverse section of a blood vessel (vein) from the vicinity of a capsule of an eight year old brain abscess. The patent lumen shows red cells and two lymphocytes; the adventitial, greatly dilated, space exhibits a vast amount of connective tissue fibers resembling a scar and containing in the interspaces numerous plasma cells, especially in the external areas. Many plasma cells are assuming the shape of fibroblasts. Use a hand lens. (Same stain as in Figure 7, × 600.)

DIRECTION OF FLOW OF BRAIN TISSUE FLUIDS

But to enable pathologic elements to reach the subarachnoid space from the depths of the brain tissue, there must be some pathway leading from the latter to the former; this pathway is composed of the perivascular spaces of Virchow-Robin. That they form such a connecting link is generally admitted, and Weed,[2] by his remarkable experimental work, conclusively proved that the flow of the perivascular contents is from the brain to the subarachnoid space. For his experiments Weed used 1 per cent. solution of equal amounts of iron

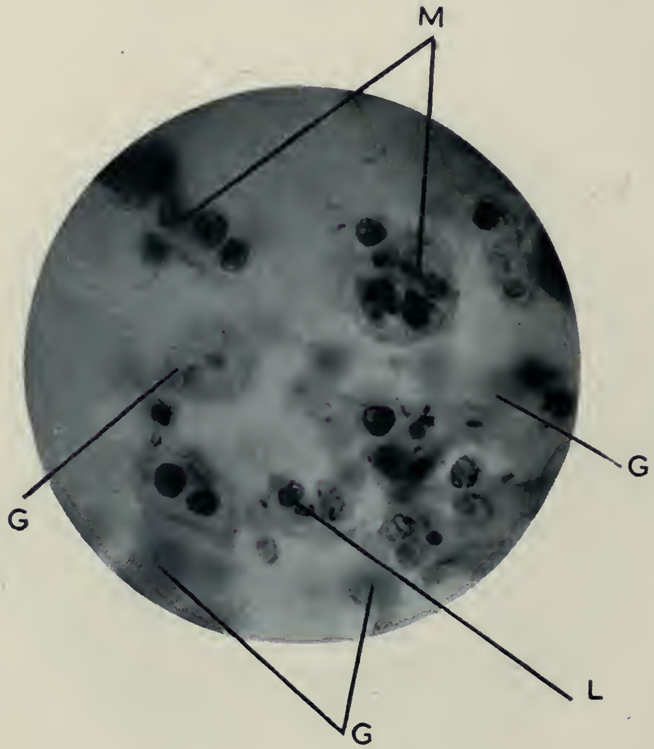

Fig. 10.—Types of infiltration cells in the subarachnoid space reproduced in Figure 8. M, macrophages; G G, Gitter cells; L. lymphocytes. (Same stain as in Figure 7, × 1200.)

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[2] Weed, L. H.: Studies on Cerebrospinal Fluid. II. The Theories of Drainage of Cerebrospinal Fluid with an Analysis of the Methods of Investigation, J. Med. Res. **26**:21, 1914-1915. III. The Pathways of Escape from the Subarachnoid Spaces with Particular Reference to the Arachnoid Villi, Ibid. **26**:51, 1914-1915; IV. The Dual Source of Cerebrospinal Fluid, Ibid. **26**:93, 1914-1915; An Anatomical Consideration on Cerebrospinal Fluid, Anat. Rec. **12**:461 (May) 1917-1918.

ammonium citrate and potassium ferrocyanid. This fluid, isotonic with the body fluids, was injected under long continued pressure of from 130 to 180 mm. of water, into the lower thoracic and upper lumbar regions, or into the arachnoid cistern about the cerebellum of cats and dogs. After the injection was completed the head of the animal was severed from the body and placed in a 40 per cent. solution of formaldehyd to which a from 1 to 5 per cent. solution of hydrochloric acid was added. The resulting precipitate of ferric ferrocyanid in the form of bluish granules (Prussian blue) could easily be identified wherever the fluid had been absorbed. The course of the absorption of the injected solution can be assumed, as Weed rightly concluded, to be the true pathway normally pursued in the absorption of the cerebrospinal fluid, as the results are obtained with nontoxic solutions practically isotonic with the body fluids. These pathways, as indicated by the deposits of the Prussian blue granules, prove to be the pial meshes, the subarachnoid space, the dural spaces, the pacchionian bodies and the sinuses (longitudinal, cavernous) as well as the perineural spaces of some cerebral nerves, diploe vessels and lymph channels (outside of the cranium, but none in the brain tissue proper). Practically the same pathways could be traced in a case of metastatic carcinoma of the cerebral meninges[3] in which, as in Weed's experiments, their invasion by carcinomatous cells was not accompanied by that of the brain. Only by using high pressure in his injections, did Weed succeed in finding the Prussian blue granules in the adventitial spaces of the brain vessels.

These experimental and pathologic facts apparently prove that the brain substance, under normal conditions, does not obtain anything from the subarachnoid space whatever its content may be—Prussian blue, cancer cells or a solution of arsphenamin. This contention is supported by Figure 11, a photomicrograph from a case of cerebrospinal meningitis. The pia is greatly infiltrated by plasma cells, the subarachnoid space is distended and separated from the arachnoid by a mass of pus cells, while the cerebellar tissue does not show a single pus cell, plasma cell or any other kind of abnormality. In some way the plasma and pia cells are kept, as it were, from penetrating into the cerebellar substance. This would not be the case had the flow been from the meningeal spaces toward the cerebral or cerebellar substance.

While this finding, together with the above mentioned results of Weed's work, seem to prove that the cerebrospinal fluid or the content of the subarachnoid space does not flow toward the brain, there are comparatively few and less convincing facts tending to show that the current of the brain tissue fluids is toward the subarachnoid space and away from the brain. These brain tissue fluids are normally present in the adventitial or perivascular spaces of Virchow-Robin, which spaces it is generally admitted, are connected with the subarachnoid space, the container of the cerebrospinal fluid. Spina's,[4] Foerster's[5] and Weed's[2] experiment's brought out some striking evidence in favor

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[3] Hassin, G. B.: Histopathology of Carcinoma of the Cerebral Meninges, Arch. Neurol. & Psychiat. **1**:705 (June) 1919.

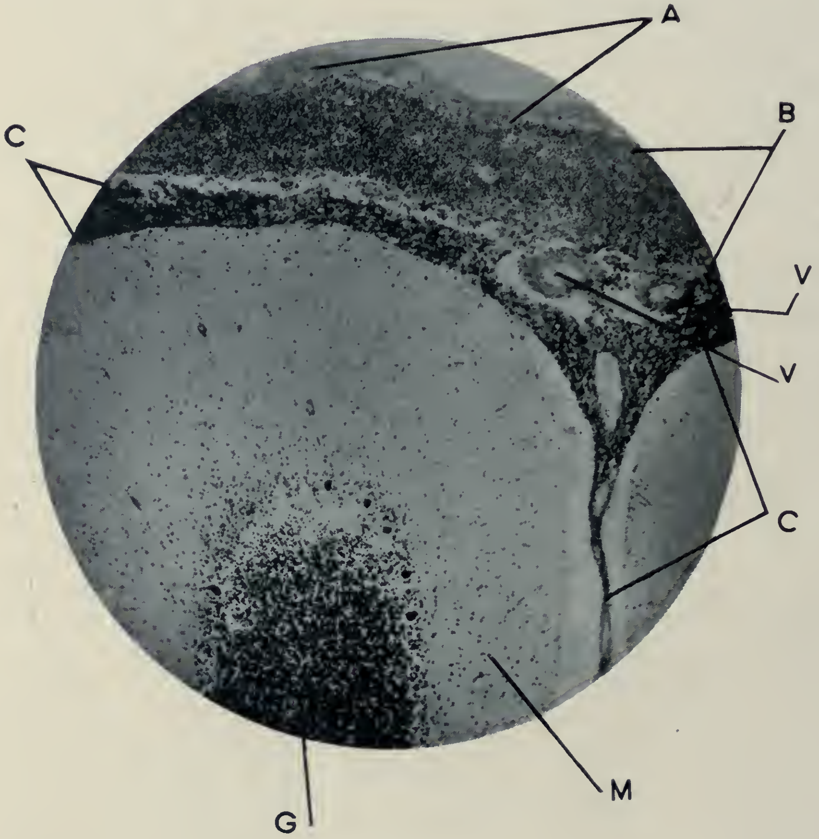

Fig. 11.—Pia arachnoid and subarachnoid space from a case of cerebrospinal meningitis. A, arachnoid; B, subarachnoid space packed with pus cells; C, pia infiltrated with plasma and mesothelial cells; V V, blood vessels of the pia with an infiltrated adventitia; M, molecular layer of the cerebellum, free from any foreign elements; G, granular layer of cerebellum surrounded by Purkinje cells. The molecular layer (M) shows two prominent capillaries with densely stained endothelial cells. (Toluidin blue stain, × 60.)

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[4] Spina, A.: Experimentelle Untersuchungen über die Bildung des Liquor Cerebrospinalis, Arch. f. d. ges. Physiol. **76:**204, 1899.

[5] Foerster, E.: Experimentelle Beiträge zur Lehre der Phagocytose der Hirnrindenelemente, Nissl's Arbeiten **11:**173, 1908.

of the opinion that the current is from the adventitial spaces toward the subarachnoid space. Spina found beads of fluid on the surface of the cerebrum after he had produced increase of pressure in the brain. Foerster found india ink granules in the subarachnoid space after injecting sterile india ink solution into the cerebral substance, and Weed, as already noted, found granules in the brain vessels when he made injections under high pressure. The pathologic findings in brain abscess conclusively show, in my opinion, the correctness of the view of the foregoing investigators that the flow is from the adventitial spaces of the brain toward the subarachnoid space. The enormous masses of fat in the capsule, as shown in Figure 5, are discharged or transported to the subarachnoid space by the way of the perivascular spaces of the cerebral vessels. The fat substance in the subarachnoid space could not reach it in any other way. Neither could the presence of fat in the perivascular spaces of the cerebral blood vessels themselves be explained, unless it came from its source of supply — the abscess and its capsule. This outward flow of brain tissue fluid seems to me to be but a natural and unavoidable process. The waste of the activity of the brain elements must be removed in some manner, and as the central nervous system lacks lymphatics, this removal is accomplished by the system of closed formations, known as Virchow-Robin's perivascular spaces. It follows that any substance injected into the brain substance will be eliminated into the subarachnoid space probably without being assimilated by the ganglion cells. On the other hand, any appropriate substance injected into the subarachnoid space will be discharged, as Key and Retzius and Weed showed, through the arachnoid villi (pacchionian bodies) into the blood stream without reaching the brain substance at all.

ABSENCE OF PHYSIOLOGIC SUPPORT FOR INTRASPINAL THERAPY

The practical conclusion is obvious; namely, that intraspinal therapy of cases in which especially the parenchyma of the brain is deeply involved, as in general paresis, has no physiologic support. In those cases in which the trouble is in the subarachnoid space itself, as in cerebrospinal meningitis, the injection is wholly justified, for given in suitable doses it may produce results. But as used in intraspinal therapy, in very small doses (fractions of a grain of arsphenamin), therapeutic results can hardly be expected. I believe that the favorable reports of the Swift-Ellis method of treatment by some reliable observers can be explained, not by the action of the intraspinal injections, but by that of the intravenous ones. Arsenic injected into the blood may reach the spinal fluid and the meninges, as demonstrated among others by G. W. Hall,[6] while Tilney[7] found the membranes stained with trypan blue after injecting it intravenously. The foregoing observations show that a drug injected into the veins may reach the meninges, and there is no object in trying to accomplish the same result by the intraspinal route.

One more point deserves attention and at least a brief discussion. It has been almost generally admitted, especially by French and English observers (Laignel-Lavastine, Carrier, Pierret, Wilkes and McEwen) that every meningitis is an encephalitis, that every inflammation of the pia is accompanied by inflammation of the brain tissue proper. This opinion has been shown to be erroneous by the histologic research work of Ranke,[8] while the foregoing considerations relative to the connections of the meningeal spaces with those of the brain, as well as to the flow of the brain tissue fluids, decidedly favor Ranke's views. Cases of pure leptomeningitis are not accompanied by encephalitis. The superficial layers of the cortex may be invaded from the meninges by pathologic elements and exhibit some inflammatory phenomena, but the latter will spare the deeper strata of the brain as well as its smallest blood vessels. If the latter are also involved, we have an encephalitis which is independently caused like the meningitis by the same morbid agent. These considerations also apply to poliomyelitis in which the parenchymatous and interstitial changes in the spinal cord are associated with meningitis. To consider the former as a product of the latter, that is, to assert with Harbits and Scheel,[9] for instance, that the pathologic changes of the spinal cord in poliomyelitis are caused by the leptomeningitis, is not in accord with the facts, for both the meningeal and spinal cord changes are due to one cause. Such, in brief, are some of the facts that can be more or less definitely established from histopathologic studies of suppurative conditions of the brain.

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[6] Hall, G. W.: Report before the Chicago Neurological Society, Feb. 19, 1920.

[7] Tilney, Fred: Long Island M. J. **8**:121, 1914; J. Nerv. & Ment. Dis. **41**:454, 1914.

[8] Ranke, Otto: Beiträge zu der Lehre von Meningitis Tuberculosa, Nissl's Arbeiten **2**:334, 1908.

[9] Harbitz, F., and Scheel, O.: Pathologisch-Anatomische Untersuchungen über akute Poliomyelitis und verwandte Krankheiten, Christiania, 1907.

FURTHER OBSERVATIONS ON THE PRESENCE OF ARSENIC IN THE SPINAL FLUID

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In 1915 one of us reported some work on this subject (G. W. H.),[1] and in 1917 Hall, Schlegel and McNally made further examinations (Marsh's test) with the following results (unpublished): In thirteen cases the spinal fluid was withdrawn twelve hours after the intravenous administration of either novarsenobenzol or neodiarsenol brand of arsphenamin in 0.6 gm. doses and in four instances the presence of arsenic was noted. In twelve cases treated similarly and the spinal fluid withdrawn twenty-four hours after the intravenous medication, no arsenic was found.

March 15, 1919, we reported to the Chicago Neurological Society a new procedure which we hoped would facilitate the passage of arsenic into the spinal fluid after its intravenous administration. We now report in full the results of this work and the method used.

PROCEDURE AND RESULTS OF ARSENICAL TESTS OF SPINAL FLUID

We chose patients who, so far as we know, had never been subjected to any form of intravenous medication except as noted in the accompanying table. Serum from the patient's blood was injected into the spinal canal and at intervals of from four to eleven hours afterward, an arsenical preparation was given intravenously. At different intervals after the intravenous injection, the spinal fluid was withdrawn and tested for arsenic. We hoped to develop a safer and more effective method than that of Swift-Ellis. The table gives our results in eighteen cases.

Mehrtens and MacArthur, who used a similar technic, report the presence of arsenic in several instances in quantities less than the practical delicacy of the test will permit. According to their table, arsenic was detected in quantities smaller than 1 micromilligram.[2]

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[1] Hall, George W.: The Presence of Arsenic in the Spinal Fluid, J. A. M. A. **64**:1384 (April 24) 1915.

[2] Mehrtens, H. G., and MacArthur, C. G.: Therapy of Neurosyphilis Judged by Arsenic Penetration of Meninges, Arch. Neurol. & Psychiat. **2**:369 1919.

Findings in Tests for Presence of Arsenic in the Spinal Fluid in Eighteen Cases

Clinical Diagnosis	Previous Arsenical Medication	Blood Wassermann Test	Spinal Fluid			Autoserum Intraspinously				Arsphenamin Injection Intravenously		First Withdrawal of Spinal Fluid				Second Withdrawal of Spinal Fluid				Third Withdrawal of Spinal Fluid			
			Wassermann Test	Globulin Test	Cell Count	Time Given	Amount in C.c.	Cell Count	Control Arsenic Test	Hours After Serum Injection	Amount	Hours After Arsphenamin Injection	Amount in C.c.	Cell Count	Arsenic Test	Hours After Arsphenamin Injection	Amount in C.c.	Cell Count	Arsenic Test	Hours After Arsphenamin Injection	Amount in C.c.	Cell Count	Arsenic Test
4/11/19 Early syphilis	None	4+	0	Not recorded 4/12/19		4/22/19 12 noon	10	360	Negative	5¼	0.6 Neodiarsenol	7½	10	4,000	Positive, trace	18½	8	2,000	Negative	40¼	8	360	Negative
4/18/19 Tabes dorsalis	None	0	4+	+	154 4/23/19	5/5/19 5 p.m.	12	110	Negative	4½	0.6 Neodiarsenol	12¾	10	1,115	Negative	22	10	280	Negative	38	10	210	Negative
5/2/19 Tabes dorsalis	None	0	0	Not recorded		5/15/19 12 noon	12	20	Negative	5	0.6 Neodiarsenol	6	10	230	Negative	41	10	74	Negative	113	10	35	Negative
5/2/19. Tabes dorsalis crisis	None	0	4+	+	56 5/2/19	5/5/19 5:30 p.m.	15	30	Negative	5	0.6 Neodiarsenol	3½	10	590	Negative	9½	10	1,260	Negative	25½	10	480	Negative
6/2/19, Cerebrospinal syphilis	None	0	4+	++	124 6/2/19	6/4/19 2:15 p.m.	12	88	Negative	3¾	0.6 Neodiarsenol	5½	8	1,110	Positive	17½	10	180	Positive	39½	10	45	Positive
4/21/19 Tabes dorsalis	++ Intravenous, Intraspinal 4/1/17	0	4+	++	165 5/9/19	6/4/19 2:30 p.m.	12	62	Negative	4¼	0.6 Neodiarsenol	5¾	5	760	Positive	17¾	8	1,070	Negative	39¼	10	80	Negative
5/9/19 Tabes dorsalis	None	0	0	++	35 5/9/19	6/4/19 2 p.m.	15	16	Negative	5¼	0.6 Neodiarsenol	5	8	1.800	Negative	17	10	2,880	Negative	39¼	10	420	Negative

5/17/19 Cerebrospinal syphilis	None	0	4+	Not recorded 5/19/19	6/4/19 1:45 p.m.	16	1,648	Negative	4¾	0.6 Neodiarsenol	5¾	8	2,070	Negative	17¾	10	2,140	Negative	40	15	2,000	Negative
6/6/19 General paresis	None	4+	4+	+ 78 6/7/19	6/10/19 4:30 p.m.	10	90	Negative	10¾	0.6 Metz Neosalvarsan	8	10	400	Negative	17½	8	1,830	Negative	35¼	10	440	Negative
5/20/19 Taboparesis	++ Intravenous, Intraspinal 6/1/18	0	4+	++ 14 5/20/19	6/10/19 4:15 p.m.	15	35	Negative	11¼	0.6 Metz Neosalvarsan	8	10	400	Negative	17¼	10	1,320	Positive	35¼	10	860	Positive
5/12/19 Cerebrospinal syphilis	None	0	0	++ 100 5/13/19	6/10/19 4:45 p.m.	10	450	Negative	11¼	0.6 Metz Neosalvarsan	8	10	1,180	Negative	17¾	10	1,240	Negative	35¼	10	180	Negative
5/26/19 General paresis	None	0	4+	+ 240 5/26/19	6/10/19 5:15 p.m.	12	280	Negative	12	0.6 Metz Neosalvarsan	7¾	10	220	Negative	17½	15	Too bloody	Negative	35¼	10	Too bloody	Negative
Tabes	None	...	...	20	10/19/19 10:30 a.m.	15	.....	Negative	6	0.6 Neosalvarsan	1	10	470	Negative	7	10	480	Negative	24	10	210	Negative
Tabes	None	...	...	11	10/23/19 9 a.m.	15	.....	Negative	6	0.6 Neosalvarsan	1	8	4,000	Negative	6	8	3,710	Negative	18	15	980	Negative
Cerebrospinal syphilis	None	...	...	66	11/6/19 9 a.m.	10	.....	Negative	6	0.6 Neosalvarsan	1	8	322	Negative	7	8	134	Negative	18	5	100	Negative
Tabes	None	...	...	6	10/16/19 11:30 a.m.	10	.....	Negative	5½	0.6 Neosalvarsan	1½	8	75	Negative	6	8	30	Negative	24	10	44	Negative
Tabes	None	...	...	10	10/16/19 10:30 a.m.	15	.....	Negative	5	0.6 Neosalvarsan	1¼	10	33	Negative	6	8	150	Negative	23½	10	200	Negative
Cerebrospinal syphilis	None	...	...	360	10/16/19 11 a.m.	8	.....	Negative	6½	0.6 Neosalvarsan	..	..	......	.....	..	..	......	.....	23½	10	......	Negative

Mr. R. Bodmer, in his discussion of John Webster's paper, on the "Excretion and secretion of Salvarsan and Neosalvarsan," stated that he had had a good deal of experience in the estimation of small quantities of arsenic in toxicologic cases, but he said that the accurate estimation of such small quantities as 0.01 mg. in 100 gm. seemed to him a rather difficult matter. Mr. Chapman remarked, in discussing the same paper, that 0.002 mg. was about the safe limit with zinc and hydrochloric acid.[3]

In our investigation, arsenic was determined in the spinal fluid by the Gutzeit method which is described in the United States Pharmacopeia and in the Journal of the Association of Official Agricultural Chemists.[4] In this method the detection of arsenic depends on the reaction of arsin with the bromid or chlorid of mercury whereby a color is produced. The color ranges from lemon yellow through orange yellow to reddish brown. Preliminary treatment of the substance in question is necessary in order to change the arsenic into an inorganic salt from which it is readily converted into arsin by means of nascent hydrogen. The change to the inorganic salt may be accomplished by strong oxidizing reagents, such as nitric and sulphuric acids, hydrochloric acid and potassium chlorate, or by bromin water in the presence of hydrochloric acid. The use of bromin as an oxidizing agent for the digestion of organic material in the determination of arsenic is suggested by C. S. Smith, in the Bureau of Chemistry Circular No. 102, 1912. In the same circular is mentioned the superiority of mercuric bromid over mercuric chlorid for the preparation of the sensitized paper used to detect the presence of arsin.

All reagents must be free from arsenic. In each case a blank must be run to make sure of this fact. We have made no attempt to determine the amount of arsenic present, but simply its presence or absence. Approximately 10 c.c. of spinal fluid are digested in a 150 c.c. flask on a steam bath for two hours with 5 c.c. of concentrated hydrochloric acid. The mixture is allowed to cool and 5 c.c. of saturated bromin water are added. The flask is then replaced on the steam bath and allowed to remain until all traces of bromin have disappeared. After cooling, the solution is transferred to a bottle and diluted with water to make about 25 c.c. In the stopper of the bottle is fitted a small piece of glass tubing in which is placed a pledget of cotton saturated with 5 per cent. lead acetate solution. Into the end of the tube is fitted a smaller tube in which is placed a strip of filter paper 4 mm. wide, previously treated with a 5 per cent. alcoholic solution of mercuric bromid. When all is ready, three drops of a 40 per cent. solution of stannous chlorid in concentrated hydrochloric acid and 5 c.c. of a 15 per cent. solution of potassium iodid are added to the contents of the bottle. Finally from 5 to 7 gm. of granulated zinc are added. As soon as the zinc is added, the stopper is quickly replaced and any change in the paper noted. The colors obtained are compared with the color obtained from a blank run at the same time and under the same conditions. Using the best reagents procurable, a slight yellow color often develops on the paper of the blank which in the hands of the inexperienced might be considered a trace of arsenic. However, if the experimental apparatus shows no deeper coloration than a blank run under the same conditions, no arsenic should be reported.

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[3] Analyst **41**:231, 1916.

[4] United States Pharmacopeia, Philadelphia, P. Blakiston's Son & Co. **9**:584. J. Assn. Official Agric. Chemists **2**:171, 1916.

The absolute delicacy of this method is set at 0.00008 mg. or 0.08 micromilligrams of arsenic trioxid.[5] The practical delicacy with bands of paper 4 mm. wide is 0.001 mg. or 1 micromilligram, that is, one part per million on a gram sample. In analytic chemical work few methods have greater delicacy than this test. The usual clinical laboratory methods seldom if ever approach its accuracy.

If this method is to be used in clinical work more than the usual precautions must be used. Special care must be taken to prevent contamination with arsenic either in withdrawing the fluid or in making the test.[1] Otherwise, arsenic may be detected in the spinal fluid derived from other sources than the blood stream. For example, arsenic determinations made in a room in which arsphenamin mixtures are prepared for injections are liable to show the presence of arsenic. In our own work we have tried to avoid any possible source of contamination with arsenic.

CONCLUSIONS

Arsphenamin given intravenously in 0.6 gm. doses may be detected in the spinal fluid in from 25 per cent. to 35 per cent. of the cases.

Irritation of the meninges by the injection of autoserum into the spinal canal does not increase this percentage.*

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[5] Autenrieth-Warren: Detection of Poisons, Ed. 4, Philadelphia, P. Blakiston's Son & Co., p. 235.

* The laboratory technic was carried out by Clayton S. Smith, Ph.D., Department Laboratory of Physiology and Pharmacology, Northwestern University Medical School.

A REPORT OF ELEVEN CASES OF CERVICAL SYMPATHETIC NERVE INJURY, CAUSING THE OCULOPUPILLARY SYNDROME

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The report of the following series of cervical sympathetic nerve lesions, due to gunshot wounds, observed in the U. S. Army General Hospital No. 11, was prompted by the comparatively large number coming under our care at one time, and by the relatively few cases noted in military literature to date. It may be that more such cases have not been recorded during the present war, first, because in the stress of work they were overlooked; secondly, because of their slightly defined symptoms; and lastly, because it is possible that no evidence of a Claude Bernard-Horner syndrome appeared until some weeks or months after injury, the patients by that time having passed from under medical supervision. Our patients, on the other hand, had in some instances been wounded from six to nine months before admission to the hospital, and sufficient time had elapsed for symptoms to develop. Then too, we had the opportunity to go over each case thoroughly, and to keep the patient under observation.

Heretofore the majority of sympathetic paralyses have occurred in civil life, and have been due to causes other than traumatic, such as cervical rib, aneurysm, enlarged glands, mediastinal tumors, and other conditions; although some have been due to injuries, and a few have been recorded in times of war. The earliest data we have on this subject, especially from the standpoint of ocular symptoms, dates back to 1727, when Pourfour du Petit [1] made his experiments on the cervical sympathetic nerve. In 1851, Budge [2] localized the center for the pupillodilator fibers between the sixth cervical and the fourth dorsal vertebrae. Claude Bernard,[3] in 1858, working on animals, observed the ocular phenomena resulting from stimulation and section of the cervical sympathetic, while Horner, in 1861, published the first clinical case of paralysis of this nerve. Mitchell, Moorehouse and Keene,[4] in charge of the nervous wards of the U. S. Army Hospital, Turner's Lane, Philadelphia, during the Civil War, gave the first accurate account of a gunshot wound of the sympathetic. Mr. Paget, in 1865, referred to this condition in connection with injuries to the brachial plexus.

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[1] Du Petit, Pourfour: Memoire sur la portion cervicale du nerf grand sympathique; Memoires de l'Academie des Sciences, 1727.

[2] Budge: Experiences demoutrant que l'origine du nerf sympathique dans la moelle epiniere, Compt. rend. Acad. d. sc. **35**:255, 1851.

[3] Bernard, Claude: Recherches experimentales sur le grand sympathique et specialment sur l'influence que le section de ce nerf exerce sur la chaleur animal, Compt. rend. Soc. de biol. **5**: Pt. 2, 277, 1853.

Coming down to the present, Roche [5] reported four cases in 1916, and collected seven others, while Burger [6] recorded one in 1917, and Tournay [7] one in 1918. Our series of eleven cases was picked from 530 cases of peripheral nerve and spinal cord injury.

It is interesting to compare the prominence of symptoms in different patients, and the completeness or incompleteness of the ocular sympathetic syndrome. This may be due, according to Bernard,[8] to the fact that the center for oculopupillary action is situated at the eighth cervical and first dorsal vertebrae, while the centers for vasomotor, secretory and trophic changes are located at the second to sixth dorsal vertebrae. A lesion may thus involve one without affecting the others. In this connection, he demonstrated by experiments on dogs, that section of the first dorsal root produced oculopupillary symptoms, while section below this point of second to fourth roots caused only vasomotor changes.

Oppenheim demonstrated in a gunshot wound of the neck, where the first dorsal segment was exposed, that by stimulating the anterior roots oculopupillary symptoms were produced, but none occurred on stimulating roots below the first dorsal.

Krause thinks there are only oculopupillary symptoms if the spinal cord is affected, while there are vasomotor symptoms if the rami communicantes are principally involved. On the other hand, Mme. Dejerine Klumpke,[9] experimenting on dogs in 1885, demonstrated oculopupillary phenomena only when rami communicantes of the first dorsal nerve were affected.

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[4] Mitchel, Moorehouse and Keene: Gunshot Wounds and Injuries of the Nerves, Philadelphia, J. B. Lippincott, 1864.

[5] Roche: Les paralysies du sympathique cervical dans les blessures de guerre, Arch. d'Ophth. **35**:339 (Nov.-Dec.) 1916.

[6] Burger: Contributions a l'etude de syndrome Claude Bernard-Horner, Arch. méd. Belges. **70**:309 (Apr.) 1917.

[7] Tournay: Remarques sur l'inegalite pupillaire dans des cas de myosis unilateral par defect sympathique, Bull. de l'Acad. de méd., Par. **80**:486 (Dec. 3) 1918.

[8] Bernard, Claude: Recherches experimentales sur le grand sympathique et specialment sur l'influence que le section de ce nerf exerce sur la chaleur animal, Compt. rend. Soc. de biol. **5**: Pt. 2, 277, 1853.

[9] Klumpke, Mme. Dejerine: Dejerine's Semiologie des Affections du Systeme Nervous, Ed. 2, 1885, pp. 610 and 1156.

It is not our purpose in this report to attempt to localize the pupillary, vasomotor or trophic fiber, but to present the cases in order of prominence of their ocular symptoms, and then to discuss them briefly from the standpoint of their neurologic diagnosis. Before doing this, however, a summary of the anatomy and physiology of the cervical sympathetic nerve might be of assistance.

ANATOMY AND PHYSIOLOGY OF CERVICAL SYMPATHETIC NERVE

The dilators and sphincters of the iris are too well known to need description, but the exact anatomy of the involuntary muscles of the orbits is still unsettled. In general, these muscles may be described as follows:

Behind the eyeball smooth muscle fibers occur in the periosteum and form a strong layer closing the orbital fissures. This is the musculus orbitalis which prevents the contents of the orbit from sinking backward. In the tendons of the levator palpebrae superiosis there are also smooth muscle fibers, and closely related to these are the tarsal muscles, small masses of involuntary muscle which lie at the bases of the upper and lower lids and insert into the tarsi. All of these, through their tonic activity, tend to keep the palpebral fissure widely open.[10]

The innervation of the pupil is double, the midbrain autonomic system sending fibers to the sphincter iridis, and the cervical sympathetic supplying the dilator. The orbital and tarsal muscles, and the nonstriated part of the levator palpebrae superioris are supplied only by the sympathetic system.

The spinal center for these sympathetic nerves lies in the lateral horn of the eighth cervical and first and second thoracic segments. Closely associated with this center are the cells of origin of the fibers that control the sweating and vasomotor reactions of the face. Some authors believe that the latter functions have their centers lower down the cord, in the second to fourth thoracic segments.[11] From these centers in the cord preganglionic fibers arise and pass via the corresponding cervical nerves and remi communicantes to the cervical sympathetic chain, finally terminating in the superior cervical ganglion.

Superimposed on the spinal center there is doubtless a cortical control, for such highly integrated reactions as fear and pleasure have their oculopupillary and vasomotor concommitants. A bulbar center also may be postulated, since lesions high in the cervical cord may cause myosis.

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[10] Landstrom: Ueber Maebus Basedowi, Thesis, Stockholm, 1907.

[11] Higier, H.: Nervous and Mental Diseases, Monograph Series No. 27, New York, 1919.

The peripheral nerves (in this case postganglionic sympathetic fibers) arise from cells in the superior cervical ganglion. These fibers at first are collected in the internal carotid plexus, but soon take separate courses. The more inferior branches probably spread out to supply the sweat glands and blood vessels of the face. The more superior branches generally follow the ophthalmic branch of the trigeminal nerve, and pass with the vasociliary nerve into the ciliary ganglion, from which they emerge and proceed with the long and short ciliary nerves to their endings in the smooth muscle fibers.

Physiologically, it must be kept in mind that the action of these nerves is tonic. In the case of the pupil there is a balance of two antagonistic systems, the autonomic sphincter and the sympathetic dilator; so the autonomic is allowed to act without restraint and cause continuous myosis if the sympathetic innervation is cut off. In the case of the vasomotor fibers to the blood vessels of the face, cutting off the vasoconstrictor control might be expected to cause flushing, but it is possible that there are also vasodilator fibers that may bring about active flushing. Here again the exact anatomy is not known so that the reactions cannot always be interpreted. Stimulation of the cervical sympathetic causes increased sweating of the face, so interruption of these fibers causes anhidrosis on the affected side. As the smooth muscles of the eyeball are normally in tonic contraction, they keep the globe forward and the lids separated. Cessation of this function is followed by ptosis, narrowing of the palpebral fissure and enophthalmos.

Due to the anatomic relations of the structures just described, certain symptoms are frequently associated with this sympathetic syndrome. In the first place, injuries to the spinal cord above or at the ciliospinal center may cause varying degrees of spastic paraplegia, quadriplegia or hemiplegia. Most common of all is the lower brachial plexus paralysis (Klumpke's paralysis) with atrophy of the small muscles of the hand and anesthesia of the dermatomes corresponding to the lowest cervical and upper two thoracic nerves. This condition indicates a lesion of the nerve roots before they enter the plexus. The recurrent laryngeal nerve may also be injured, causing hoarseness. In addition any number of traumas may be found, such as fractures of the vertebrae, injury to the larynx, etc. It is strange that any of the patients with these bullet wounds of the neck survived.

REPORT OF CASES

Case 1.—*History.*—Private, Co. K, 30th Inf.; aged 23. Previous to the injury the history was unimportant. There was no suspicion of syphilis. Oct. 11, 1918, at Verdun, the patient was struck by a piece of shell in the left side of the neck just above the midclavicle; the missile lodged in the soft tissue just to the left of, and slightly above, the spinous process of the third dorsal vertebra. There was immediate paralysis of the left arm, and the patient stated that for a week he saw double. By the first week in November he could move the shoulder, arm and forearm. Sensation also began to return except in the distribution of the eighth cervical root. At about this time the wound healed. Early in December voluntary motion returned at the wrist. He was admitted to U. S. Army General Hospital No. 11, on Nov. 30, 1918.

Examination.—Voluntary motion at the left elbow was weak and restricted. There was complete loss of flexion and extension of the fingers of the left

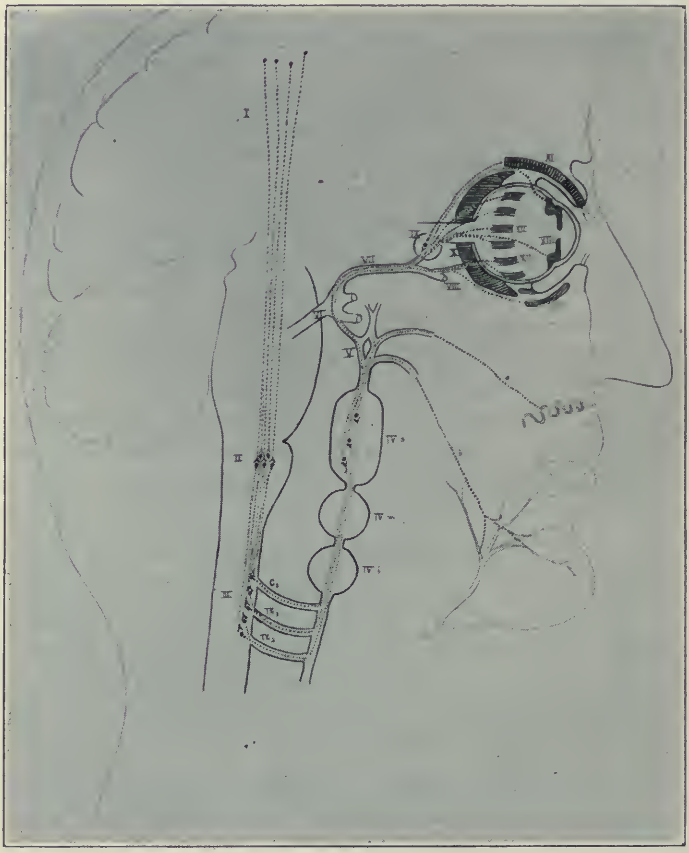

Fig. 1.—I, cortical path; II, bulbar center; III, ciliospinal center in lower cervical and upper thoracic cord, C. 8, Th. 1 and Th. 2 represent rami communicantes to cervical sympathetic chain; IV, cervical sympathetic ganglions, inferior, middle and superior; V, internal carotid plexus, giving off branches to (a) sweat glands of face, and (b) to blood vessels of face; VI, trigeminal nerve; VII, ophthalmic nerve; VIII, nasociliary nerve; IX, ciliary ganglion; X, orbital muscle (of Müller); XI, levator palpebral superioris with sympathetic branch to the smooth muscle fibers; XII, tarsal muscles; XIII, dilator muscles of the pupil. (N. B. The drawing is diagrammatic. No attempt is made to show the exact anatomic relations.)

hand, and only slight extension of the wrist. The entire extremity showed atrophy; the fingers were stiff and cyanotic. Sensory examination showed anesthesia in the distribution of the whole brachial plexus; but by January 23 this hypesthetic area was practically normal. With massage and electrical treatment there was marked improvement during the next three months, but no return of voluntary motion in the intrinsic muscles of the hand. Myosis, enophthalmos and ptosis of the left eye were noticed by the patient from the beginning. Unilateral anhidrosis was present on the left.

Diagnosis: Contusion of the whole left brachial plexus, with severance of the seventh and eighth cervical roots.

Ophthalmologic examination revealed marked enophthalmos, ptosis and myosis of the left side; left palpebral fissure 10 mm.; right, 13 mm; both pupils reacted to light, accommodation and consensually, but the left slower than the right. The diameter of the left pupil was 2.5 mm., right 3.5 mm.; under cocain, left 2.5 mm., right 5 mm. Tension, left eye 13 mm. of mercury, right 17 mm. of mercury; no heterochromia irides. Vision 20/20 in both eyes; near point left 11 cm., right 12 cm. Ocular movements normal. Left globe slightly elevated and left nictitating membrane slightly more prominent than right. Left cornea appeared a little flatter than right. On excursion of the eye to the right, the difference in the size of the pupils increased slightly, while on turning to the left the difference diminished slightly. Fundi and fields were normal. No flushing of the face or hemiatrophy was noted. Lacrimation was normal.

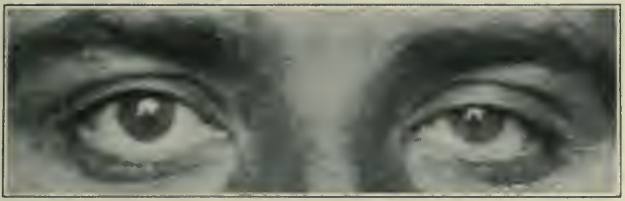

Fig. 2 (Case 1).—Marked enophthalmos, ptosis and myosis of the left eye.

Case 2.—*History.*—Private, Co. L, 18th Inf.; aged 24. The past history was negative. He did not have syphilis. At Soissons, July 21, 1918, he was struck by a piece of high explosive shell in the right forearm and was thrown about 5 feet by the explosion, landing on his right shoulder. The right arm immediately became numb and paralyzed. At Evacuation Hospital No. 5, he was operated on and the bones of the right arm were set. No ether was necessary on account of the anesthesia of the arm. During the next three months there was no improvement in the paralysis. He was admitted to U. S. Army General Hospital No. 11, Oct. 24, 1918.

Examination.—This revealed that on the outer side of the right forearm there was a scar 1.5 inches long. The right arm was completely paralyzed, and there was restricted motion of the shoulder. Electrical examination shows normal response to faradic stimulation in the trapezius and supraspinatus and infraspinatus muscles; all the other muscles of the right upper extremity failed to respond. With the galvanic current there was a slow wavy contraction in all the muscles that failed to respond to the faradic, except the intrinsic muscles of the anterior surface of the hand in which there was no response. Sensory examination showed anesthesia in the distribution of the fifth, sixth, seventh and eighth cervical and first thoracic nerves.

On Nov. 14, 1918, an operation was performed by Lieut.-Col. Charles H. Frazier. The posterior triangle of the neck on the right was explored and a bulbus enlargement was found that represented the remnants of the brachial plexus. It extended from the clavicle upward, tapering off into a single trunk which proved to be the fifth cervical. The retracted stumps of the sixth, seventh and eighth cervical nerves could not be found, although search was made almost up to the intervertebral foramina. After this operation the patient stated he first noticed that his right pupil was smaller than the left. It is probable that the oculopupillary symptoms were present immediately after the injury, but passed unnoticed; and that their onset had no relation to the operation. A second operation was performed on Dec. 19, 1918, by Lieut.-Col. Frazier. The exposure was similar and the fifth cervical nerve root was identified and grafted with two strands of the musculocutaneous nerve to a nerve in the shoulder, which was thought to be the circumflex.

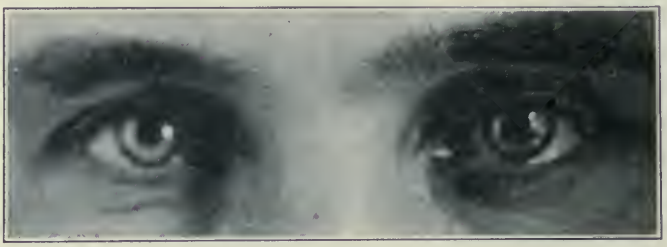

Fig. 3 (Case 2).—Inequality of pupils before instillation of cocain.

Diagnosis. Evulsion of the sixth, seventh and eighth cervical nerve roots and of the first thoracic root of the right brachial plexus with contusion and fibrosis of the fifth cervical root.

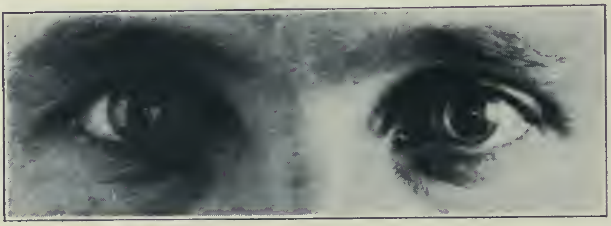

Fig. 4 (Case 2).—Showing increase of inequality after instillation of cocain.

Ophthalmologic Examination.—This revealed marked enophthalmos, ptosis and myosis of the right side; right palpebral aperture 9 mm., left 12 mm.; pupillary reactions normal, but right slightly retarded; diameter right pupil 2.5 mm., left 6 mm.; width under cocain, right 2.5 mm., left 9 mm.; ocular tension, right 12 mm. of mercury, left 16 mm. of mercury; vision 20/15 both eyes. Near point, right 9 cm., left 10.5 cm.; right eye slightly elevated; ocular movements normal. On excursion of the eyes to the right the difference in the size of the pupils diminished, on excursion to the left the difference in the size increased slightly. Fundi and fields were normal. The patient showed a slight hemiatrophy of the right side of the face.

Case 3.—*History.*—Private, Co. C, 58th Inf.; aged 27. The past history was negative. On October 5 he was struck by a piece of shrapnel while advancing at Verdun. This missile entered in the midline anteriorly just below the larynx and passed out of the lateral aspect of the neck through the trapezius muscle. There was immediate paralysis of the left arm and aphonia. Two weeks later his voice and the movements of the left hand began to return. A week later he noticed that his left eye was sensitive and that the lashes fell out. From this time on there was steady improvement. He was admitted to U. S. Army General Hospital No. 11 on Dec. 31, 1918.

Examination.—Physical examination showed paralysis of the left deltoid and supraspinatus and infraspinatus muscles, with weakness of the brachicardialis, biceps and triceps. There was no sensory disturbance. The voice was hoarse and laryngoscopy showed partial paralysis of the left vocal cord. The left palpebral fissure was narrower than the right and the pupil contracted. During the next four months there was improvement in the voice and the arm movement, but the scapular muscles and deltoid were still atrophied, and weak.

Diagnosis: (1) Contusion of left brachial plexus with a severe lesion of the fifth cervical root, causing a more permanent scapulohumeral type of paralysis; (2) partial paralysis of left recurrent laryngeal nerve; (3) interruption of left cervical sympathetic nerve.

Ophthalmologic Examination.—Marked enophthalmos, ptosis and myosis of left eye. Left palpebral aperture 7 mm., right 10 mm. The pupils reacted normally, the left being slightly slower than the right; width of left pupil 2.5 mm., right 3.5 mm.; under cocain, left 2.5 mm., right 5 mm.; tension of left eyeball 23 mm. of mercury, right 25 mm. of mercury. The left cornea appeared slightly flatter than the right. The vision of the right eye was 20/20, left 20/20; near point, left 11 cm., right 13 cm. Ocular movements were normal. The left eye was slightly elevated. There was slight diminution of relative size of pupils on gazing to extreme left, with a slight increase in size on directing gaze to the right. Fundi and fields were normal. There was no disturbance of lacrimation or of vasomotor functions.

Case 4.—*History.*—Private, Co. D, 47th Inf.; aged 30. The past history was negative. No syphilis was present. On July 30, on the Champagne front, he was struck three times — first by a piece of shrapnel in the neck entering just to the left of the thyroid cartilage and passing out behind the sternomastoid muscle at the same level; second, by a machine-gun bullet from behind, which fractured the first thoracic vertebra and lodged in the soft parts, being removed the next day; and third, he received a gutter wound in the biceps of the right arm. His legs were stiff and weak, and he was unable to walk. He could not speak and the left arm was paralyzed. During September his voice returned, but was hoarse. In late October he could walk a few steps if supported.

Examination.—Examination on admission to U. S. Army General Hospital No. 11 revealed that the whole right side of the body below the third rib was anesthetic to pain and temperature stimuli. In the distribution of the eighth cervical and first and second thoracic nerves on the left there was anesthesia to all forms of cutaneous stimulation. The arm was carried partly flexed, and motion was limited at wrist and elbow; the thumb and fingers were spastic and weak with fibrosis of the joints. The muscles of the trunk were hypertonic and the tendon reflexes were greatly exaggerated with bilateral clonus. Babinski's sign was elicited on both sides by touching the patient anywhere below the third rib, and even by jarring the bed. Electrical examination showed that all muscles reacted to the faradic current. There was hyperhidrosis of the right side of face. The left eye showed ptosis, enophthalmos and myosis. Roentgen-ray examination revealed a fracture of the first thoracic vertebra.

Course and Treatment.—On November 5 laminectomy was performed by Lieut.-Col. C. H. Frazier, and the cord was decompressed at the eighth cervical and first and second thoracic vertebrae.

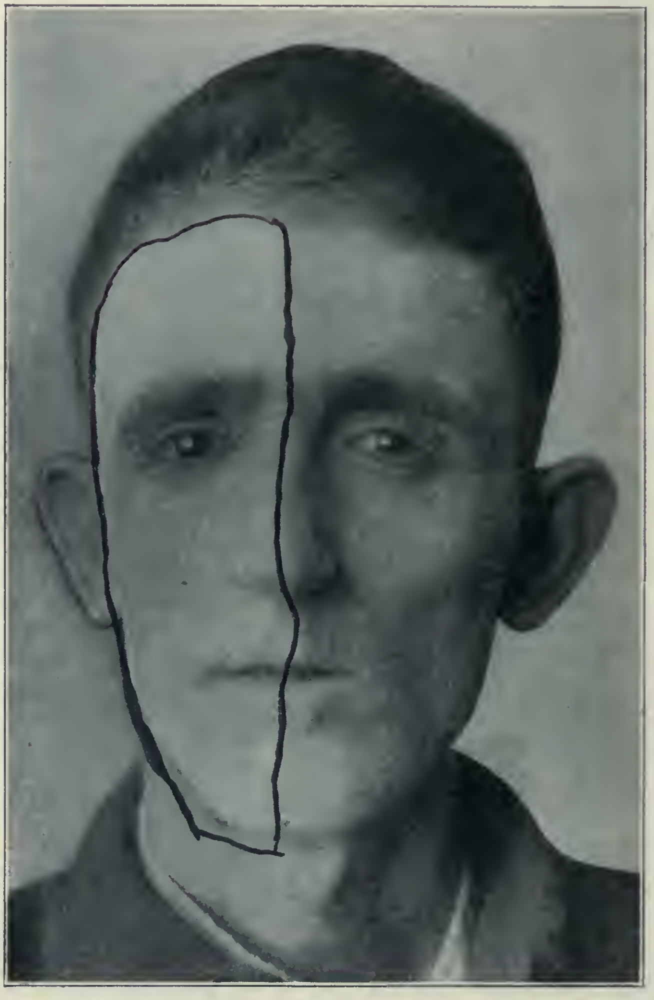

Fig. 5 (Case 4).—Showing unilateral sweating of the face.

Three months later the patient was able to walk half a mile. Anesthesia on the right had disappeared, but it was still present in the left arm. The oculopupillary and sudomotor symptoms persisted. The tendon reflexes were still exaggerated, and Babinski's sign could be elicited on both sides, but the clonus had disappeared. The left arm was more freely moveable, but the fingers were still spastic and weak.

Diagnosis: (1) Contusion of the spinal cord at the level of the eighth cervical and first and second thoracic segments, from machine-gun bullet. (2) Interruption of the left recurrent laryngeal nerve, and of the left cervical sympathetic nerve by a piece of shrapnel.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis of left side, less marked than in previous cases; left palpebral fissure 11 mm., right 13 mm.; pupillary reactions normal, but left slower than right; diameter left pupil 2.5 mm., right 3.5 mm.; under cocain, left 2.5 mm., and right 4.5 mm. Tension was the same in both eyes. Vision was 20/20 in both eyes. Ocular movements were normal in all directions. No change was noted in the relative size of the pupils when looking to the right or the left. The fundi were normal. There was no disturbance in lacrimation; but marked flushing on the left side of the face.

Case 5.—*History.*—Corporal, Co. F, 26th Inf.; aged 20. The past history was negative; there was no history of syphilis. At Soissons on July 19, 1918, the patient was struck in the right side of the neck by a machine-gun bullet that entered 2 cm. above and 4 cm. to the right of the thyroid cartilage and lodged to the right of the vertebral column at the level of the fifth and sixth cervical vertebrae. Four days later it was removed at operation. There was immediate paralysis of the right arm and shoulder, which began to improve after a few weeks.

Examination.—On admission to U. S. Army General Hospital No. 11, on August 27, motor disability was limited to weakness of the right arm and hand, with inability to flex the thumb and index finger. There were sensory disturbances in the area of distribution of the seventh root; light touch and pain were poorly felt, and there was pain and tenderness in the radial half of the forearm and hand. Electrical examination showed that all muscles responded to the faradic current. Narrowing of the right palpebral fissure, with enophthalmos and myosis was conspicuous.

During October there was marked improvement, and late in December he was transferred to a Convalescent Detachment.

A special eye examination, December 10, showed: Vision, right 20/15, left 20/15. External examination: Slight ptosis of right upper lid. Right pupil 3 mm. in diameter, left 4.5 mm., both reacted to light and accommodation. Ocular movements were good in all directions. Ophthalmoscopy: Fundi negative.

Diagnosis: (1) Contusion of the right brachial plexus causing temporary complete paralysis of the right arm, with a residual irritative lesion of the seventh cervical root, causing symptoms for six months; (2) interruption of cervical sympathetic nerve in the neck.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis of right side. Width of right palpebral fissure was 8 mm., left 10 mm. Pupils reacted to light and accommodation. Diameter of right pupil was 3 mm., left 4.5 mm.; under cocain, right 3 mm., left 7 mm. Ocular movements were good in all directions; fundi negative; vision, right 20/15, left 20/15. There was unilateral anhidrosis on right side.

Case 6.—*History.*—Private, Co. G, 28th Inf.; aged 20. The past history does not reveal syphilis or other important facts. On July 18 he was struck in the right posterolateral aspect of the neck by a machine-gun bullet which passed forward and downward, coming out through the thyroid cartilage slightly to the right of the midline. There was immediate paralysis of this shoulder and upper arm, with aphonia for two weeks, and hoarseness thereafter. On August

21 the oculopupillary symptoms were noted. On August 27 there was no return of motor function and hypesthesia was found on the right arm in the distribution of the seventh cervical nerve. On October 3 he was admitted to U. S. Army General Hospital No. 11.

Examination.—The examination here showed paralysis and atrophy of the right scapulohumeral muscles, with marked atrophy and no response to faradic stimulation, except weak contractions in the triceps and biceps. There was no sensory disturbance. Myosis, enophthalmos and ptosis were present on the right, with unilateral anhidrosis. There was gradual improvement, and on February 13 he was discharged with voluntary movement and faradic response in all muscles. The oculopupillary symptoms did not improve.

Diagnosis: (1) Contusion of the outer cord of the right brachial plexus causing paralysis of the right arm and shoulder of the scapulohumeral type; (2) interruption of the right cervical sympathetic nerve in the neck; (3) interruption of the right recurrent laryngeal nerve.

Ophthalmologic Examination.—Enophthalmos, ptosis and myosis were present on the right. The right palpebral aperture was 6.5 mm., left 8.5 mm. Pupillary reactions were normal; pupillary measurements not recorded; vision, right 20/50, left 20/20. Refractive error was present in the right eye; fundi negative; unilateral anhidrosis.

Case 7.—*History.*—Private, M. G. B., 9th Inf.; aged 28. On July 1, 1918, at Chateau Thierry, he was wounded by shrapnel in the neck and right arm. The projectile entered the neck 5 cm. above the junction of the inner and middle thirds of the clavicle and lodged against the spinal column, fracturing the transverse process of the seventh cervical vertebra.

For the first month he had pain in both arms, paralysis of both legs, and of the right arm, and weakness of the left arm. Bladder and rectal control were poor for six weeks. There was steady improvement and by March 6, when he was admitted to U. S. Army General Hospital No. 11, he could walk stiffly.

Examination.—The examination showed a Brown-Séquard paralysis with loss of pain and temperature sense on the left side below the seventh cervical segment, and weakness and spasticity of the right leg. There was also a partial musculospiral nerve lesion of the right arm. Myosis of the right pupil and "hemisweating" were noted on July 6. At operation on April 15, 1919, a fragment of shell was removed from the right trapezius muscle.

Diagnosis: Contusion of right side of the spinal cord at the level of the eighth cervical segment.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis of the right side; width of right palpebral fissure 10.5 mm., left 12 mm. The pupils reacted normally to light and accommodation; size of right pupil 2.5 mm., left 3 mm. Under cocain, right 3.5 mm., left 6 mm. Ocular tension, right 21 mm. of mercury, left 23 mm. of mercury; vision, right 20/20, left 20/20. Muscular excursions were normal. On turning the eyes to the extreme right there occurred a slight diminution in difference between the size of the two pupils, while the opposite occurred when looking to the extreme left; fundi negative. There was anhidrosis of the right side of the face.

Case 8.—*History.*—Private, Co. B, 11th M. G. B.; aged 29. The past history was negative. Syphilis was denied by name and symptom. On Sept. 28, 1918, while on duty in the Argonne Forest, he was struck by a piece of aerial bomb, which entered the neck in the midline anteriorly and did not emerge. The left arm was immediately paralyzed completely and the legs were weak.

There was also bleeding from the mouth and inability to speak. A week later voluntary motion began to return in the fingers, and since that time there had been steady improvement. The legs regained their motor power in two months. There was difficulty in controlling the bladder for two months. No eye symptoms were noticed by the patient. He was in bed most of the time until his admission to U. S. Army General Hospital No. 11, on Dec. 5, 1918. He could then walk a short distance, and by January 1 could walk a mile.

Examination.—Physical examinations made at various times from December 5 until March 17 and in various special departments revealed: Tachycardia constant between 110 and 130; the heart was otherwise normal. There was a small circular wound scar on the anterior surface of the neck in the midline over the larynx. Voluntary motion was weak at the left shoulder, elbow and wrist. There was atrophy of the left arm, forearm and hand. The skin of this forearm and hand showed scaliness and the nails were long and tender. There was hypohidrosis in the left hand. The tendon reflexes were exaggerated on the left, and in the left leg there was a short patellar clonus and a positive Babinski sign. The whole right side of the body showed a hypesthesia to pain and temperature below the level of the tenth dorsal segment. The left vocal cord was fixed near the midline. The left palpebral fissure was narrower than the right. There was pain in the left shoulder and hand, and the muscles of the left arm were tender, especially the triceps.

Diagnosis: (1) Contusion of the spinal cord in the region of the eighth cervical segment, more on the left than on the right side; (2) contusion of the sixth, seventh and eighth roots; (3) interruption of left recurrent laryngeal nerve.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis of the left eye; left palpebral fissure 9 mm., right 11 mm. Pupils reacted to light and accommodation, but the left was slightly slower than the right. Diameter of the left pupil was 3 mm., right 3.5 mm.; under cocain, left 5.5 mm., right 7 mm.; ocular tension, left 16 mm. of mercury, right 17 mm. of mercury; vision, left 20/20, right 20/20 uncorrected; left near point 12 cm., right 16 cm. No abnormal change in the relative size of the pupils was apparent on turning the eyes to the extreme right or left. The left nictitating membrane appeared slightly more prominent than the right. Fundi and fields were negative. Slight diminution of sudomotor activity was noted on the left side of the face.

Case 9.—*History.*—Private, Battery I, 6th Field Artillery; aged 31. The past history was negative—no syphilis. On June 4, 1918, at Cantigney, the patient was struck in the back of the neck with a piece of high explosive shell that fractured the sixth cervical vertebra and lodged in the vertebral canal. He was immediately completely paralyzed in all four extremities. About July 15 voluntary movement began to return in both hands and arms, and a month later in the left leg. There was no movement in the right leg until November. There was complete loss of bladder and rectal control until October, when he began to have slight improvement. He was admitted to U. S. Army General Hospital No. 11, on Nov. 6, 1918.

Examination.—Physical examination showed hypalgesia below the second rib on the entire left side. The right palpebral fissure was narrower than the left. There was motor weakness and spasticity in all four extremities; the movement of the right leg was limited to flexion of the toes, the right arm was weaker than the left.

Course and Treatment.—Treatment was directed to overcome spastic contractures and decubital ulcers. On January 23 a laminectomy was performed by Lieut.-Col. Charles H. Frazier, and a fragment of shell was removed from the dura at the level of the junction of the sixth and seventh cervical vertebrae; it was pressing on the right posterior aspect of the cord. In the next three months there was slight improvement in the movement of the legs, due largely to physiotherapy, but the patient was still in bed with bad contractures of the legs. Bladder control improved immediately after operation so that he could hold his urine for nearly two hours, instead of fifteen minutes as was the case just before operation.

Diagnosis: Compression of the seventh and eighth cervical segments of the cord, mainly on the right side, causing a partial Brown-Séquard syndrome and narrowing of the right palpebral fissure.

Ophthalmologic Examination.—This revealed enophthalmos, ptosis and myosis on the right side. Right palpebral aperture was 10 mm., left 11.5 mm. The pupils reacted to light, accommodation and consensually. Slight difference in activity between the two was noted, the right being a little slower than the left. Diameter of the right pupil was 3.5 mm., left 4 mm.; under cocain, right 4.5 mm., left 8 mm.; tension, right eye 18 mm. of mercury, left 22 mm. of mercury; vision, right eye 20/20, left eye 20/20; right near point 10 cm., left near point 12 cm. The right eye was slightly elevated from the normal position. The fundi were normal.

CASE 10.—*History.*—Private, Co. I, 9th Inf. The past history was negative, and there was no evidence of syphilis. On July 18, 1918, at Soissons, the patient was struck by a machine-gun bullet. At the time he was aiming at a machine-gun nest in a tree, and he thought the bullet came from that gun and struck him in the left cheek, ranged downward and came out just at the left of the spinal column. He was immediately paralyzed in all four extremities, but in two weeks he could move his legs and right arm freely. During the next four months there was steady improvement, and in November he could walk a mile.

Examination.—In April, 1919, the positive findings were: Fracture of the fifth, sixth and seventh cervical vertebrae shown by the roentgen ray; stiffness and limitation of motion in left hand and wrist; tendon reflexes increased in arms and legs, less in left arm than in other extremities; muscular spasms in arms and legs occasionally. Constipation was present; there was no bladder disturbance, but there had been loss of libido and potentia since the injury.

Diagnosis: Contusion of the spinal cord in the region of the seventh and eighth cervical segments.

Ophthalmologic Examination.—Enophthalmos and myosis were present on the left side. No ptosis was noted. The width of each palpebral fissure was 10 mm. The diameter of the left pupil was 3.5 mm., right pupil 5 mm. The pupils reacted to light, accommodation and consensually; activity of the left was slightly diminished. Tension of the left eye was 20 mm. of mercury, right eye 25 mm. of mercury; vision, left eye 20/20, right 20/20; near point of left eye 13 cm., of right eye 14 cm. Muscular excursions were normal; fundi normal.

CASE 11.—*History.*—Corporal, Co. E, 125th Inf.; aged 23. On Oct. 5, 1918, he was struck by a machine-gun bullet which grazed the right shoulder and passed into the right side of the neck and out of the left side of the neck at a slightly higher level. Aphonia was the only symptom noticed immediately, and after a few weeks he was able to whisper hoarsely.

Examination.—Examination, made November 5, showed immobility of the right vocal cord. On Feb. 7, 1919, this cord moved slightly and the voice was less hoarse. A slight narrowing of the left palpebral fissure was present; there was no myosis or unilateral sweating. (See special ophthalmologic examination.) Routine neurologic examination elicited no spinal cord symtoms. He was discharged on February 27, with improvement in his voice.

Diagnosis: (1) Partial paralysis of the right recurrent laryngeal nerve, recovering; (2) partial interruption of the left cevical sympathetic nerve. This diagnosis seems probable, because the symptoms do not correspond to those found either in the group of cord contusions or of nerve lesions presumably complete.

Ophthalmologic Examination.—Enophthalmos, ptosis and slight narrowing of palpebral fissure were noted on the left side. No myosis was present. The left palpebral aperture was 11 mm., the right 12 mm. Both pupils reacted normally to light and accommodation. Each measured 3 mm. in diameter, and under cocain the dilatation was the same on both sides. Vision in both eyes was 20/20. Ocular movements were normal in all directions. The fundi were negative.

Findings and Diagnosis in Authors' Cases

Case	Enophthalmos	Ptosis	Myosis	Hypotension	Palpebral Fissure	Pupil	Pupil (Cocain)	Vision	Near Point	Unilateral Anhidrosis	Vasomotor Disturbance	Hemiatrophy Facialis	Heterochromia Irides	Diagnosis
1	+++	++	+	+	13 10	3½ 2½	5 2½	20/20 20/20	12 11	+	—	—	—	R. Root severance 7, 8 cervical
2	+++	++	+	+	9 12	2½ 6	2½ 9	20/15 20/15	9 10½	+	+	+	—	R. Root evulsion 6, 7, 8 cerv. 1st th.
3	+++	++	+	+	13 10	3½ 2½	5 2½	20/20 20/20	13 11	—	—	—	—	Interruption of L. cerv. sympathetic nerve
4	+	+	+	—	13 11	3½ 2½	4½ 2½	20/20 20/20	12 11	+	+	—	—	Interruption of L. cerv. sympathetic nerve
5	+	+	+	—	8 10	3 4½	3 7	20/15 20/15	..	+	—	—	—	Interruption of R. cerv. sympathetic nerve
6	+	+	+	—	6½ 8½	.. ..	.. ..	20/50 20/20	..	+	—	—	—	Interruption of R. cerv. sympathetic nerve
7	+	++	+	+	10½ 12	2½ 3	3½ 6	20/20 20/20	9 9	+	—	—	—	R. cord contusion, C. 8
8	++	+	+	—	11 9	3½ 3	7 5½	20/15 20/20	16 12	—	—	—	—	L. cord contusion, C. 8
9	+	+	+	+	10 11½	3½ 4	4½ 8	20/20 20/20	10 12	—	—	—	—	R. cord compression, C. 7, 8
10	+	—	+	+	10 10	5 3½	7 5	20/20 20/20	14 11	—	—	—	—	L. cord contusion, C. 7, 8
11	+	+	—	—	12 11	3 3	.. ..	20/20 20/20	10 10	—	—	—	—	Partial interruption of L. cerv. sympathetic nerve

SUMMARY

In correlating the above data, the most striking fact is shown by arranging the cases in the order of the severity of their ocular symptoms, and then observing the grouping of the neurologic diagnoses.

Cases 1 and 2, the most severe, are both root lesions. They are followed by four cases of lesions of the cervical sympathetic nerve. All of these showed more conspicuous eye phenomena than the following four cord contusions. The last case had mild symptoms and seemed to be a nerve lesion, but the neurologic examination was unsatisfactory, and there is a possibility that this case was one of cord contusion, or partial interruption of the cervical sympathetic nerve.

Reviewing separately the more important symptoms, these results are presented:

1. Enophthalmos was the most constant symptom, being present in all cases, but most marked in the two root lesions, with a diminishing degree of prominence as we go down the scale through the nerve injuries, until the cord contusions are reached, where in Cases 10 and 11 it was only faintly perceptible.

2. Only one case failed to show at least a moderate degree of ptosis, and this was one of cord contusion.

3. Miosis was present in all except Case 10 (the neurologic examination of which, as we above mentioned, was unsatisfactory). It is interesting that the pupils of the cases with the more severe lesions, namely the root and nerve injuries, failed to dilate under cocain, while the cord contusions dilated, but to a reduced degree, the range being from 1 to 2 mm.

4. Hypotension was present in only five of eleven cases — the two root and three contusion injuries. The nerve lesions showed no difference in tension.

5. In comparing the width of the palpebral apertures, we find that the greatest difference between the sound and affected sides is seen in Cases 1 and 2 (root lesions) and Case 3 (the most severe nerve injury). Each of these shows a difference of 3 mm. The other nerve cases register a difference of 2 mm., while three of the four cord lesions present a variance of 1.5 to 2 mm.

6. Sudomotor disturbances were recorded in over 50 per cent. of our cases, and as with the other symptoms, unilateral anhidrosis was present in the more severe lesions, namely, Cases 1, 2, 4, 5, 6 and 7.

7. Only two patients showed vasomotor disturbances, Cases 2 and 4. The explanation for this may be that we did not see our patients until four to six months after injury, and possibly symptoms that existed immediately afterward had disappeared.

8. Heterochromia irides was not present in any one of the eleven cases examined. Presuming that this condition is due to paralysis of the cervical sympathetic nerve, this symptom might be expected in the cases classified under root lesions, and the more severe nerve injuries, in which complete interruption was undoubtedly present.

Wilson[12] reported a case in 1917 in a girl 14 years of age, whose condition had existed for twelve years, resulting from swollen cervical glands. Calhoun[13] has recently reported four cases, and found only thirty-three others recorded in the literature. In all of these, heterochromia had existed since infancy or youth. After an exhaustive study, he says that heterochromia irides has never been seen in adults, as the result of sympathetic paralysis from trauma. Bistis[14] thus far has reported the only case occurring in adults, following paralysis of the cervical sympathetic nerve. As all the cases here reported were adults, in whom the paralysis had been caused by gunshot wounds, the statement of Calhoun seems to explain the absence of heterochromia irides in this series.

9. Hemiatrophy facialis was observed to a slight degree in one patient, Case 2, a root evulsion, involving the sixth, seventh and eighth cervical and first thoracic roots. Weisenberg, in an unreported case seen in U. S. Army General Hospital No. 3, saw hemiatrophy in a similar wound, six months after injury, but as in the above, it was not pronounced. Possibly if we were to observe these patients several months longer, this condition would develop in a larger number, as it has been observed quite constantly in paralysis of the cervical sympathetic of long standing.

10. Vision was normal or better in all patients but one, in whom there was a refractive error corrected to normal. An interesting point was noted in the difference between the near points of the good and affected sides. Nearly all showed a variation of from 1 to 4 cm.

11. According to Tournay,[15] the relative difference in size between the miotic and normal pupils diminishes on directing the eyes to the affected side, while the opposite is true when the eyes are turned toward the healthy side. This we found to be true to a slight degree in a few cases, but it was not a constant symptom.

CONCLUSIONS

1. Lesions affecting the seventh and eighth cervical and first thoracic roots, through which sympathetic fibers run, cause the most severe and typical oculopupillary syndromes.

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[12] Wilson: Heterochromia Irides in Nerve Case, J. Nerv. & Ment. Dis. **1**:438, 1917.

[13] Calhoun: Causes of Heterochromia Irides with Special Reference to Paralysis of the Cervical Sympathetic, Am. J. Ophth. **2**:255 (April) 1919.

[14] Bistis: La paralysie du sympathique dans l'etiologie de l'heterochromie, Arch. Ophth. **32**:578, 1912.

[15] Tournay: Remarques sur l'inegalite pupillaire dans des cas myosis unilateral par defect sympathique, Bull. de l'Acad. de méd. Par. **80**:486 (Dec. 3) 1918.

2. Lesions involving the cervical sympathetic nerves, with complete or partial interruption, produce symptoms less severe, as a rule, than root injuries.

3. Contusions of the cord result in the least pronounced phenomena, and are more prone to complete recovery than the first two conditions.

4. Heterochromia irides did not occur in adults following injury to the cervical sympathetic nerve.

5. The pupils of the cases with root and nerve injuries did not dilate with cocain; those of the cord contusions did.

6. The near point of the affected eye was less than its fellow in over 50 per cent. of the cases, the average difference being slightly over 2 cm. It is difficult to draw any definite conclusion on this point, as the greatest variation was in a case of cord contusion, with only slight ocular symptoms.

EXPLANATION OF PLATE I

Fig. 1 (Case 1).—Arrow shows point at which shell struck neck.

Fig. 2 (Case 2).—Arrow shows point at which blow was received when patient landed on right shoulder. Shaded portion shows area of sensory disturbance.

Fig. 3 (Case 3).—Arrow shows point of entrance and exit of missile.

Fig. 4 (Case 4).—Arrows show path of shrapnel and point of entrance of machine-gun bullet at first thoracic vertebrae. Shaded portions are areas of sensory disturbance.

Fig. 5 (Case 5).—Arrow shows point of entrance of machine-gun bullet near thyroid cartilage. Shaded portion is the area of sensory disturbance.

Fig. 6 (Case 6).—Arrow shows path of machine-gun bullet. Shaded portion shows area of sensory disturbance.

Fig. 7 (Case 7).—Arrow shows point of entrance of shrapnel. Shaded portion shows area of sensory disturbance.

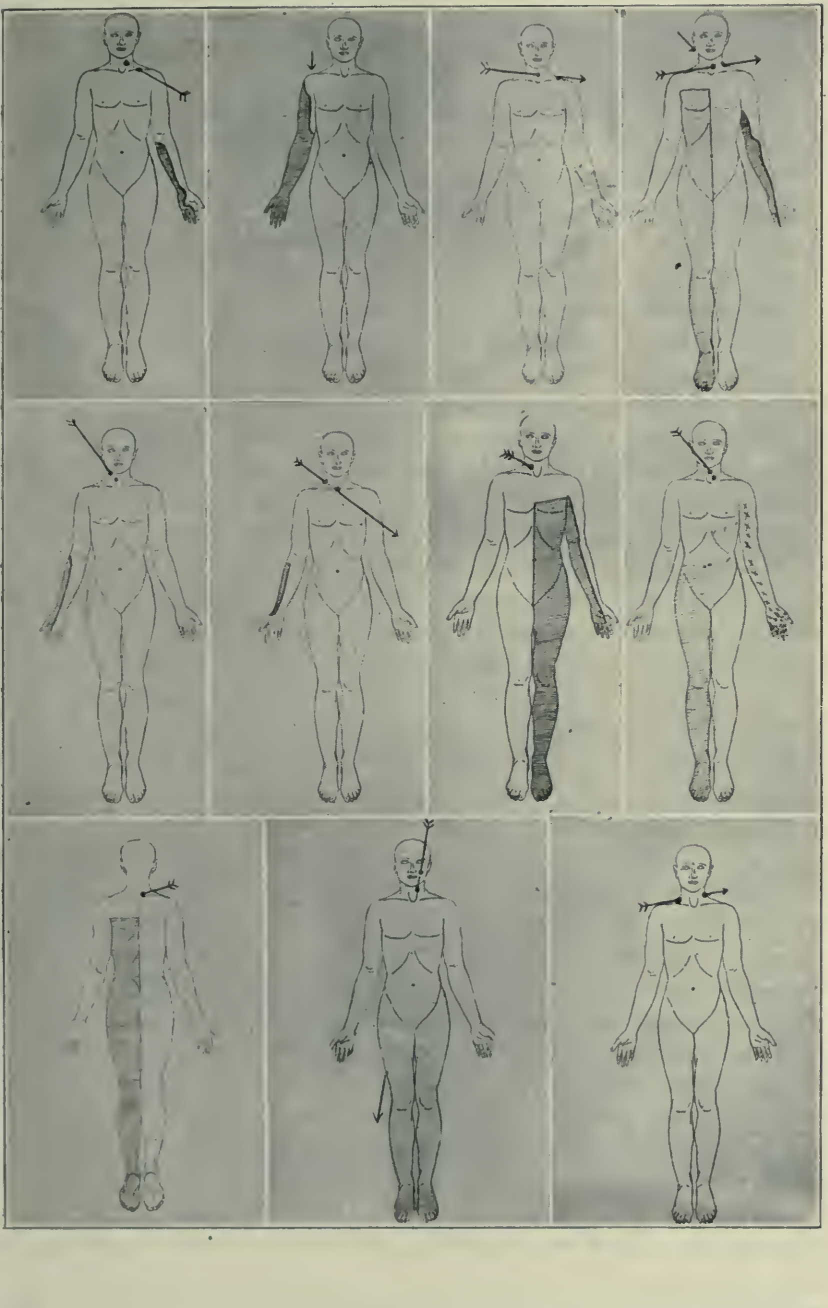
Fig. 8 (Case 8).—Arrow shows point of entrance of aerial bomb. Crosses on left arm indicate area of atrophy; shaded portion shows area of hypesthesia to pain and temperature.

Fig. 9 (Case 9).—Arrow shows point in neck at which high explosive shell struck. Shaded portion is area of hypalgesia.

Fig. 10 (Case 10).—Arrow shows points of entrance and exit of machine-gun bullet.

Fig. 11 (Case 11).—Arrow shows path of machine-gun bullet.

PLATE 1



MEDITATIONS ON MORALE*

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The war has produced many new words or usages of old words. Among such old words is one that has taken on meanings and a familiarity that did not exist in prewar days, namely, morale. According to former usage, morale has been defined as a state of mind pertaining to courage, valor and enthusiasm, used in relation to bodies of soldiers or sailors. Such a definition can hardly be called a definition, as it does little to advance one's knowledge concerning the meaning of the term.

Of course it is difficult to define the abstract in concrete terms, but the following definition of morale may be considered. Morale is a state of mind or consciousness conducive to the control of conduct by intellectual ideals rather than by instinctive impulses. In other words, when the emotional element in a person's consciousness becomes dominant and his conduct is governed by instinctive reactions to his emotions instead of by intellectual ideals he has lost part of his morale.

To some this definition may prove unsatisfactory, because it may be thought that morale is more properly a collective term having an element of cooperation for a given end. The definition, however, without including such a meaning does not necessarily exclude it.

It is interesting to consider the various great instincts that have been studied in their effect on human conduct. Bergson with his élan vital, and Jung with his life force seem to consider the instincts only as subdivisions of one essential power—the source of our conscious and unconscious strivings. Adler has stressed the impulses of self-assertion and self-abasement; Freud, the sex impulse; Trotter, the gregarious or herd impulse; and Sidis, the instinct of self-preservation and the emotion of fear.

To discuss the trends of modern clinical psychiatry, or to champion the importance of the rôle of one instinct rather than another as the all important catalizer of human conduct—normal and psychopathic—is to step off into deep and by no means quiet water. Of the various instincts mentioned, it is evident that two — the gregarious and the self-preservation instincts—have come forward especially in war time, and have entered strongly into the building up and also into the breaking down of morale.

When the instincts and the intellect have worked in harmony, we have usually found mental health, activity and euphoria; when the instincts and the intellect have been in conflict, we have seen cacophoria, hesitation and often ill health. Should we buy liberty bonds or pay our debts; should we stick to our home job or enlist? The conflict of the herd instinct calling us to do as our colleagues were doing, and the intellectual decision to do something else, is still vivid in our minds, and last November marked the end of more than one conflict.

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*Read at the October meeting of the Boston Society of Psychiatry and Neurology.

The factors conducive to the augmentation of the instinctive reactions may be considered under three heads: (1) inheritance, (2) acquired disabilities, and (3) environmental strain.

Inheritance influences morale along one or both of two lines. In the first place, unquestionably there is of course vast variation in congenital physical conformation and development, and strength along the more subtle physical characteristics that are defined as immunity to disease. Some persons are handicapped by being born bleeders; some persons, on account of their physical conformation, are predisposed to certain mechanical difficulties such as malnutrition from a sagging stomach or headache from focusing eyes that are inclined to squint. Others are poorly started in life—they are premature infants or are born of sick mothers. Such congenital physical disabilities unquestionably increase the burden borne by many in their struggle to live successful and happy lives. Such disabilities, however, by no means necessarily lessen morale. In many cases the person by a sort of compensatory process develops exceptional ability along unhandicapped lines and makes a resulting success of life which often surpasses that of the physically well endowed.

Another way in which congenital handicaps may lessen morale is through fear of following in the footsteps of parents, which predisposes to the development of ills and handicaps from which the parents suffered. Many an individual explains his bad temper on the ground that his father had a bad temper, or attributes his indulgence in alcohol to a similar parental weakness, or in explanation of a headache says that his mother had the same kind of headache. If mental disease has shown itself in a parent, a patient may be under constant dread of the appearance of a similar condition in himself. Dread of developing weaknesses and conditions exhibited by a previous generation undoubtedly is often an important factor in the inception of a conscious or subconscious fear, and such a fear is a definite source of lowered morale.

Acquired disabilities either of a temporary or permanent character tend to reduce morale. They do this in the first place by reducing physical strength, and thus increasing the difficulties that must be surmounted to gain success. The greater the difficulty with which success is attained, the more likely an individual is to fear failure in the attainment of success; or, in other words, the more easily is the instinct of self-preservation and its accompanying fear, emotion, aroused. In the second place, acquired physical disability may render a person more sensitive to emotional strain and also more predisposed to react excessively to such emotional strain. These excessive reactions gain the attention and in turn become secondary objects of fear and apprehension.

Acquired disabilities, especially if permanent or of long standing, may cease to be a cause of lowered morale and the person may develop along unhandicapped lines exceptional ability tending to unusual success.

Environmental strain caused many of the war neuroses, and here, as many writers have pointed out, the instinct of self-preservation and the emotion of fear have been the most important factors.

The work of the morale division of the army has been mainly to foster and augment intellectual ideals, and to remove or mitigate as far as possible the conditions that beget ill health, anxiety and fear. It has extolled the reasons for the entry of America into the war; it has encouraged self-confidence and developed esprit de corps, furthered pride in the various army divisions and preached the invincibility of American troops. It has encouraged every agency that combated discomfort and homesickness and has glorified the importance and necessity of personal purity. Poor food, poor shelter, lack of occupation, poor organization, petty rivalries and jealousies among men and officers and physical illness—both venereal and nonvenereal—lessened morale. Doughnuts, songs, sports, letters from home, decorations for valor and other encouragements, by lessening emotional strain and augmenting intellectual ideals helped keep the morale high.

If we could plot out on a chart our intellectual ideals and our instincts, would we not find that our morale would be represented by the separation between the two curves? With fall in ideals, or with a rise of instinctive reactions, the margin of morale decreases. And when fear of the Hun, or of personal death or injury, or when loss of property, hunger, sex desire, mob activity or race hatred rise above intellectual ideals, is not morale gone? Without morale an army is ready for defeat and rout. Without morale, an individual is ready to develop a psychoneurosis.

Abstracts from Current Literature

ABSTRACTS OF SOME RECENT WORK ON THE NEUROGLIA BY THE SPANISH SCHOOL

(*Continued from page 552*)

HISTOLOGICAL AND HISTOPATHOLOGICAL INVESTIGATIONS ON THE PINEAL GLAND IN MAN. N. Achucarro and J. M. Sacristan, Trab. d. lab. de invest. biol. Univ. de Madrid **10**:185, 1912.

The authors give a summary of the bibliography, treating especially the work of Dimitrowa (**2**:3, 1905, of Studuicka [Jena] Cutore, Bizzozero, Le Neuraxe), Meynert, Hagemann, Henle, Cionini, Weigert, Cajal, Krabbe and others.

The authors use these stains: toluidin blue, hematoxylin-eosin, Van Giesson, Alzheimer, Mallory, Weigert, Bielschowsky, Heidenhain and Cajal.

1. Connective tissue passes from the pia mater along the vessels, forming an adventitial sheath to vessels thus furnishing adventitial lymph spaces as in the vessels of the brain. This space is more dilated in pineal of regressive character. In such cases the vessels are tortuous and form pockets as in atrophic conditions of the brain. The connective tissue separates the vessels from the parenchyma and also forms strands dividing the organ into lobules and lobes of irregular size. The lobules are not always well limited.

Four types of connective tissue organization are seen: (*a*) Trabecula not forming a network, but passing simply from one point of connective tissue sheath to another; (*b*) fine network of fibers; (*c*) coarse network of trabeculae complicated in arrangement; and (*d*) many serpentine filaments, tendril like, recalling the appearance of elastic fibers (not seen in ox or sheep). These fibers resemble ovals and knots described by Cerletti in senile atrophy and are observed most in pineals which show atrophy or other marked regressive phenomena.

In some parts of the gland there is no appearance of lobulation, the connective tissue forming a thick network. In the perivascular spaces of the pineal of both man and beast mast cells are seen. Plasma cells are noticed in man and in ox, sometimes in groups, but no lymphocytes are seen with them. Granular cells are seen in the pineal of man, containing yellowish or green pigment, in cases where other marked regressive phenomena are present, tortuous vessels, dilated lymphocytes, retraction of parenchyma, etc. Pineal "sand" is seen in perivascular spaces.

The cells of the lobules are mostly nerve cells with vesicular nuclei, the membranes of which are folded and the nuclei contain frequently the so-called "Dimitrowa balls" which, in the authors' estimation, are due to foldings and inclusions of the nuclear membrane (many illustrations are shown elucidating the process). These inclusions are not always spherical, but frequently oval, oblong, etc. Bonfiglio has described like cells in severe degenerative brain disease. These cells with inclusions are much more plentiful in old age and in regressive conditions in the pineal such as connective tissue proliferation, etc. The authors, therefore, consider these "balls of Dimitrowa" as products of regression of the nuclei and not evidences of glandular secretion.

Glia cells are found in the lobules and a rich plexus of neuroglial fibers. A few medullated nerve fibers are seen, but many more nonmedullated fibers. In the periphery of the lobules and in the perivascular spaces cells are seen with many prolongations terminating in buds around the vessels. These cells contain fibrils in their protoplasm and appear to be sympathetic nerve cells. The authors' later researches show that these "end feet" and many branched fibers are proliferative changes in a cell reacting to degenerative changes in the neighborhood as demonstrated by many granular cells, etc. Connective tissue cells are seen sparsely in the lobules near the trabeculae, etc.

CONTRIBUTION TO THE KNOWLEDGE OF THE NEUROGLIA OF THE HUMAN CEREBRUM. S. Ramon y Cajal, Trab. d. lab. de invest. biol. Univ. de Madrid **11**:255, 1914.

The author concludes that:

1. Fibrous astrocytes and protoplasmatic astrocytes both originate in epithelial cells or spongioblasts of the medullary tube. They represent the results of the adaptation of an identical primitive type to two different environments. Therefore, the mesodermic origin of the glia claimed by certain authors is untenable.

2. During embryonic life and after birth the dislocated epithelial cell and astrocyte are capable of proliferation in normal circumstances.

3. The protoplasmatic astrocyte has a structure recalling that of glandular cells: in the midst of a dense spongioplasm gliosomes or differentiated granules lie. Their number, size and staining reactions vary according to functional states.

4. The interstitial net in the gray matter of cortex described by many authors is not neuroglial in nature. The ramifications of the glia form a very complex plexus.

5. Every astrocyte of both gray and white substance is provided with a suction tube or perivascular foot. This is delicate in the protoplasmatic and robust in the fibrous astrocyte.

6. All astrocytes of both man and beasts, without exception, possess a centrosome. In young animals the endocellular apparatus of Golgi is also seen. Both are seen in the area of maximum protoplasm.

7. A small adendritic body exists in the nervous centers, probably mesodermal in origin, certainly neither belonging to nervous nor to glia tissue.

8. Aside from other possible varieties of the above cell, two types are seen in all likelihood of different activities—the perineuronal or satellite type, probably the homologue of the subcapsular elements of the ganglions, and the apolar cell of the white substance, probably corresponding to the Schwann's cell.

9. The adendritic cells of the white substance may comprehend diverse physiologic categories of the mesodermal elements. Among the adendritic cells remnants of astrocytes in involution probably are present.

10. During the epoch of evolution astrocytes are capable of phenomena of migration and expansional transformation which are similar to the ameboid movements of leukocytes. In this way vascular feet are formed which sometimes are the result of a new expansion, at others of the displacement and increase in a radial or primordial prolongation.

11. The Ranvier-Weigert fibers result from the intraprotoplasmatic differentiation of the astrocytes. In no case do the fibers completely free themselves from the glial protoplasm.

12. The human cortex differs from that of animals, not only in the enormous quantity of the glandular type of cells which it contains, but also in their small size, the great richness of the interstitial glial plexus and the absence of any tendency in the glandular type of glia to differentiate intraprotoplasmatic fibers in normal conditions.

CONTRIBUTION TO THE STUDY OF THE NEUROGLIA OF THE CEREBELLUM. J. Ramon y Fananas, Trab. d. lab. de invest. biol. Univ. de Madrid **14:**163, 1916.

The author uses original Cajal fixation, sections from 20 to 25 microns placed from four to six hours at 18 to 20 C. in gold-sublimate bath. Achucarro's tannin silver shows the molecular layer, but not others.

The molecular layer of the human cerebellum shows protoplasmatic astrocytes of four types: (*a*) mono-radiated, sometimes twin; (*b*) bifurcated villous type, mostly near the Purkinje cells; (*c*) protoplasmatic type, many ramifications, generally in about the middle of the molecular layer; (*d*) perivascular astrocyte—rather rare; it has protoplasm concentrated in upper part of the nucleus, is vacuolated, has one large branch ascending giving off many fine short branches laterally and one short thick branch which lies along a capillary. Glial feet are seen only in class *d*. Autonomous and polarized cells show ependymal origin. In the rabbit there are many glial feet. Epithelial or forked cells or Bergmann's fibers are seen in all animals and man, starting in the Purkinje layer. Their nuclei are pale purple with black granules. The cell is vacuolated and has two or three long branches with more or less protoplasm. Dislocated epithelia cells are found in all parts of the molecular layer. Satellite cells of the molecular layer, both astrocyte and adendritic (nonglial) are found. The glia of the granular layer consists mostly of transitional cells, one portion of the branches is protoplasmatic, other portions are fibrous. Some fibrous astrocytes are found near the Purkinje cells.

The neuroglia of the white substance is fibrous in type, somewhat larger than in other portions of the brain. Many adendritic cells are seen.

Conclusions: 1. Molecular layers contain autonomous astrocytes, Bergmann's fibers and prolongations from cells in the granular layer. Most astrocytes of class *d* insert at least one expansion on a vessel; they contain many gliosomes. 2. About the Purkinje cells both fibrous and protoplasmatic astrocytes are found; also certain cells applied to the neuronic dendrites which may serve as insulators. 3. Adendritic cells are frequent in all parts. 4. The protoplasmatic cells contain gliosomes.

VARIATIONS IN THE HISTOPATHOLOGY OF THE NEUROGLIA IN CEREBRAL SOFTENING. P. del Rio Hortega, Trab. d. lab. de invest. biol. Univ. de Madrid **14:**1, 1916.

The author treats in great detail the regenerative types of glia seen in and about a softening: hyperplasia, hypertrophy of cells and fibers, "monster" forms of cells (certain types at first progressive later become regressive), vacuolar degeneration of dendrite masses: segmentation of the prolongations in the form of rings, rackets, clubs, etc.

Regressive phenomena coincide in time with progressive. Noted among the regressive were klasmatodendrosis beginning at the periphery and extending to body of cell and the formation of "füll Körperchen."

In studying granular cells the author has never seen anything that would lead him to infer that the glia took any part in their formation; on the contrary, he has seen many transitional forms between the adendrites or third element and the granular cell. He considers the adendritic cell the predominating type of granular cell and recognizes in addition only cells from the blood vessel in the same capacity. He distinguishes the adendritic cell and its transitional forms up to the true granular cell by the richness of its nucleus in chromatin—the neuroglial cell nucleus is poor in chromatin.

The author considers the great difference of opinion of different observers in regard to the granular cell due to the nonrecognition of the adendritic cell as a nonglial body.

ALTERATIONS OF THE CENTRAL NERVOUS SYSTEM IN A CASE OF PARALYTIC FORM OF DISTEMPER IN A DOG.

Paul Rio Hortega, Trab. d. lab. de invest. biol. Univ. de Madrid **12:** 97, 1915.

The author reviews all the work done on the subject up to date and then gives the results of his case studied by the methods of Cajal and Bielschowsky for neurofibrils and axis cylinders, by Nissl's toluidin blue and Mann's stain for the chromatic portion of the nerve elements, by Weigert's and Mallory's glia stains and Achucarro's glial and connective tissue stains, by Unna-Pappenheim's stain, toluidin blue, and iron-hematoxylin for inflammatory elements, and by Herxheimer's stain for disintegration products.

His conclusions are:

1. In distemper of the paralytic form an inflammatory process exists in the central nervous system that may be designated as a diffuse meningoencephalitis.

2. This process is characterized by foci and plaques of cellular infiltration specially localized in the gray matter of the cerebrum in connection with meningeal inflammation and in the white matter of the cerebellum.

3. The lesions of the cerebellum precede those of the cerebrum and in the latter superficial lesions precede the deeper.

4. Vascular dilatation exists in early foci of infiltration. Dilatation of the adventitial lymph spaces, increase in the adventitial elements and large accumulations of small lymphocytes and plasma cells and some polynuclear leukocytes are always present in the foci.

5. Newly formed connective tissue is present in the oldest foci and in the diffusely infiltrated areas of the cerebellum. This connective tissue invades the nervous tissue and forms a complicated network in whose meshes various classes of cells are lodged (lymphocytes, plasma cells, neuroglia and ameboid).

6. The nervous elements undergo regressive phenomena consecutive to the presence of inflammation and the presence of the causal agent of the disease.

7. The above-mentioned regressive phenomena consist, in the cerebrum, of atrophy and marginal vacuolization of the cortical cells, retraction and pyknosis of the nuclei, hypertrophy and multiplication of the nucleoli and dilatation of the medullated fibers. In the cerebellum, like degenerations are seen in the

Purkinje's cells, formation of knobs and club-shaped bodies by retraction of the axons, retraction and enlargement of the pericellular nets and vacuolization and breaking of the fibers of the white substance.

8. Parallel to the cellular destruction in the cortex there is new formation of glial elements specially of the glial fibers, with abundance of astrocytes and some rod cells.

9. Preameboid elements are plentiful in the cerebellum as are also phagocytic ameboids acting on the destroyed axons.

10. In some ganglion cells in the base of the brain and in the peduncles very small intraprotoplasmatic and intranuclear bodies are seen which show a similarity to the smallest forms of protozoa studied by Noguchi in rabies and to the "Staupe Körperchen" described in distemper by Lentz, Standfuss and Sinigaglia.

11. Although the inflammatory lesions in distemper coincide with those of rabies, the reactive phenomena of the nerve cells to the respective viruses is not identical.

12. In relation to general paralysis of the insane, a great similarity of the inflammatory process and of some secondary results, such as the connective tissue sclerosis, is seen.

DENDRITIC RAMIFICATIONS, NEWLY FORMED, IN NERVE CELLS, AND ALTERATIONS IN THE NEUROGLIA IN THE SENILE DOG. Gonsalo R. Lafora, Trab. d. lab. de invest. biol. Univ. de Madrid **12**:39, 1914

The pyramidal cells in Ammon's horn of the senile dog present numerous lateral ramifications of their dendrites, numbering twenty or more, of greater or lesser length, but in general ending not far from their point of origin. These ramifications are not seen on all the dendrites, but generally on the principal dendrite shortly after leaving the body of the cell.

This alteration is seen in small foci, yellowish in color (in silver preparations). Under high power an area is seen without intercellular fibrils, but filled with a yellowish homogeneous substance in the midst of which are seen two or three neuroglial nuclei and the dendrite from which the newly formed ramifications originate. As a rule the new ramifications do not surpass the yellowish zone. The ramifications show numerous neurofibrils by Bielschowsky's method. The ramifications are tortuous, show irregular enlargements and often terminate in bud form.

Nuclear stains (toluidin blue, etc.) do not stain the homogeneous yellowish substance, and the glia around it shows no proliferative reaction. The author considers the dendritic new-formations as a reactive or regenerative reaction similar to the formation of senile plaques. The cause of this reaction is excretion from the nerve cell.

Senile plaques in the vicinity of blood vessels are frequent and explained by the action of the lymph flow to the vessels carrying the irritating substance with it.

The irritating substance is shown to be of neuronic origin because plaques are encountered only in the cellular portions of cerebrum or cerebellum, in other terms, only where nerve cells exist, and because plaques are never found in other portions of the nervous system to which their formation could be ascribed (neuroglia, axis cylinders, etc.).

Certain alterations of the dendrites somewhat similar to those described by the author have been seen in the spinal ganglions in senility (Cajal, Trabagos **4**: 1906), and in the dendrites of the Purkinje's cells after traumatism (Cajal, Trabagos **9**:1, 1911) and in the Purkinje's cells in general paralysis (Ruiz de Arcaute, Bull. de la Soc. Española de Biologia [Jan.] 1912).

The neuroglia in senile degeneration in the dog shows marked changes seen in the hypertrophy of the glia cells and the thickening of the cellular processes.

SOME OBSERVATIONS ON THE HISTOLOGY OF THE HYPOPHYSIS (HUMAN). F. Tello, Trab. d. lab. de invest. biol. Univ. de Madrid **10**:145, 1912.

A. Anterior Lobe.—The epithelial cells of the anterior lobe contain a network of fibers stained only by methods that stain in the same manner the mitochondrias and the ergastoplasm (Bonin, Da Costa). These fibrils are not stainable by any method that stains the neurofibrils. The theory is that they represent various stages of function. Nerve cells with one, two or three dendrites and large nuclei are seen.

A variable number of nerve fibers are seen, sometimes many, sometimes few, but always in determined regions, especially close to the upper surface, to the posterior lobe and to the anterior pole. They pass along the capsule, pierce it and enter the glandular substance either passing along the connective tissue or going freely among the cells and ending usually in balls or globular distentions. They frequently throw off branches as they go and sometimes are lost by the fineness of their branching. Some fibers approach the rare nerve cells, above spoken of, and are probably the sympathetic fibers from the carotid plexus described by Luschke.

B. Posterior Lobe.—The author has never been able to find nerve cells by any methods or at any age from infancy to senility. Nerve fibers in great fascicles enter by the peduncle, separate from time to time to give passage to tortuous blood vessels, and these fibers, the vessels and glial fibers fill the whole space of the peduncle (the tortuous vessels suggest senile atrophy). On reaching the lobe these fascicles separate and the separate nerves divide forming one of the richest nerve plexes in the nervous system. All these fibers are nonmedullated. These fibers are so complicated that the author has never been able to trace their endings.

Mesodermal Tissue and Neuroglia: There are two types of cells (1) glia cells: star shaped with large nucleus which is spherical, elliptical or ovoid; abundant protoplasm prolonged into abundant ramifications; (2) the second cells are large and fusiform with a rod-shaped nucleus and protoplasm accumulated at the poles—rod cells and fibroblasts. The amount of connective tissue around the vessels and between them is very considerable.

C. Intermediate Lobe.—In infancy and childhood this is usually nothing more than a cleft lined by columnar epithelia, separating the two lobules. In later life this cleft forms many cysts. These vesicles vary in lining, but the cells are mostly flattened and polymorphous, and in several layers. Colloid material and sometimes granular is seen in the vesicles. Some cells appear ameboid and are phagocytic.

Some bipolar cells are seen, probably sensory cells. There are a moderate number of nerve fibers; they form ramifications around certain ovoid epithelial cells.

D. Pathologic Modifications of Hypophysis.—Fibers are seen in degeneration; fragmentation of a fiber takes place into a series of spheres which gradually lose all structure and become pale and finally almost invisible. All stages are seen, some showing axon and retracted fibrils, etc. These degenerated fibers are seen at all ages and in all stages of health, but in the infant and healthy adults they are scattered and distinct whereas in cases of softenings, hemorrhage, neoplasms, etc., they form foci. In old age basophil cells invade the neurohypophysis and act as neoplasm, crowding and destroying the fibers.

Regeneration of the nerve fibers takes place to a limited degree, e. g., the fibers sprout, terminal arborizations take place in unaccustomed places, masses of fibers occur, such as are seen in newly-forming nerves, the neurofibrils are separated and individualized, etc.

Granular Cells Present in Pathologic Conditions: Two varieties at least are to be noted — the ordinary active phagocyte of Merzbacher, round, ovoid or slightly elongated with coarse spherical granules and cells of the spindle-shaped variety, the nuclei of which become so obscured by the large irregular granules that they have the appearance of cylinders not resembling any cell form. Many of these cells are parallel with the vessels or with connective tissue lines. Such cells are seen isolated in youth, but increase progressively with age, just as degenerative phenomena take place in the nerve cells and fibers, and in old age these granular cells are plentiful and grouped into foci. In the case of invasion of the posterior lobe by the basophilic cells, the granular cells are in great numbers, and are seen grouped around the foci of invasion. The same thing is observed in hemorrhages, softenings, etc. The author saw large quantities of them in an idiot of 25 years and in a puerperal patient.

The author considers the second cells to be the passive variety of phagocytes described by Merzbacher, originating in the glia cells and particularly engaged in taking up the products of nerve fiber degeneration.

Gurd, Ann Arbor, Mich.

LA FONCTION PSYCHO-MOTRICE D'INHIBITION ETUDIEE DANS UN CAS DE CHOREE DE HUNTINGTON. ESSAI D'APPLICATION DES TECHNIQUES DE LA PSYCHOLOGIE EXPERIMENTALE A LA NEURO-PSYCHIATRIE. Raoul Mourgue (Villejuif, Seine), Schweiz. Arch. f. Neurol. u. Psychiat. **5**:70, 1919.

Considering the number of monographs that have been devoted to the study of divers psychoses one is struck by the paucity of investigations on the psychic aspect of chronic chorea. The discovery of definite encephalic lesions in chronic chorea by Pierre Marie and Lhermitte places this malady definitely among the organic diseases of the brain.

The author had occasion to study a patient with chronic chorea of three years' standing, who exhibited the usual neurologic manifestations seen in this disease. He had in addition a right supranuclear facial paralysis, the significance of which is not explained. It might be said that shortly before his admission to the hospital he was in an automobile accident in which he lost consciousness. His face was mask-like even during violent outbursts of temper, which he frequently exhibited; the only complaint in addition to the choreoathetotic movements was a "thickening" of the tongue. The oculocardiac reflex was absent.

The principal mental features consisted in marked difficulty in calculation, inability to describe accurately objects which he had seen, particularly if this involved spatial localization, and a mild psychosis with a persecutory trend. The writer insists that in this case, at least, the dementia was more apparent than real, since the principal difficulty was in recalling, rather than in a complete loss of facts; the inability of the patient to put these into service betrayed an interference with so-called intellectual autoconduction. If one insisted or tried to aid mental imagery, for example, by asking the patient to make a diagram of the Place de la Concorde, he could do this with remarkable accuracy. Another characteristic trait was the irritability, which was dependent on a transformation of character, also an expression of this loss of autoconduction.

The author adheres to the point of view which ascribes psychophysiology to the functions of the central nervous system, in contradistinction to the theory of W. James. Pagano has shown that chemical excitation of the anterior and middle thirds of the head of the caudate nucleus, particularly the internal portion of it, will in dogs provoke emotional phenomena which present all the characteristics of fear. Excitation of the posterior third of the same nucleus gives rise to an ensemble of phenomena attributable to anger. He refers to the conception which looks on the central gray nuclei as an apparatus for vasomotor control, although this has not been definitely demonstrated to be the case in man. He quotes Brouwer to the effect that the caudate nucleus subserves the functions of the sympathetic system, particularly in its action on the smooth muscles, while the lenticular nucleus is related to reflex movements of the striated muscles.

Chronic chorea involves, with a remarkable predilection, the frontorolandic area and the corpus striatum. Lesions of the fibers are of particular importance; the tangential, the supraradial and the intraradial fibers showing degeneration. In the frontal area there is an absolute disappearance of tangential fibers; in the rolandic area this change is marked; while in the parietal and occipital areas the fibers appear normal. The subcortical white matter, while it appears normal superficially, is in reality somewhat degenerated. Normally the cerebral cortex inhibits and controls the subcortical motor centers. Lesions, therefore, that involve the corticostriate and corticothalamic fibers permit the subcortical centers to rise to uncontrolled activity. The writer again refers to the experiments of Pagano, and feels that this explains in a satisfactory manner the transformation of character seen in chronic chorea. The irritability is more or less automatic and is endogenous in origin. The mechanism of attention is likewise essentially motor, the degree of concentration being inversely proportional to the quantity of active external diffusion by muscular activity.

The writer reports a number of experiments undertaken with the purpose of showing the inhibitory effect of the corticothalamic system. In the first of these he makes a tracing, with the aid of Marey tambours and a revolving drum, of the horizontal, vertical and sagittal movements of the arms. The patient was commanded to inhibit the movements as much as possible at certain intervals. Absolutely no evidence of inhibition was noted. The writer concludes that the incoordination of movement is the result of lesions related to the corpus neostriatum—that is, the caudate nucleus and the putamen, the functions of which are supposed to be inhibiting and coordinating.

A second experiment as made by recording the patellar reflexes, the patient being asked to resist the movement. Here, too, there was no evidence that the patient was able to comply. The same thing occurred in commanding the patient to inhibit the movements of the eyelids. Tracings were also made of the respiratory movements, but gave no positive proof that the patient was able to arrest these movements on command. He then applied the inhibition test of Patrizi, in which the plethysmograph is applied to the hand and changes in volume recorded. The patient is then told to remain as quiet as possible, while a loud noise is made by striking a violent blow on a plate; the test is then repeated, but without warning the patient of the coming noise. It was interesting to note that the patient in both of these tests exhibited absolutely no evidence of vasomotor excitation, which is so striking in normal persons. The same thing occurred when the patient was reminded of the loss of 105 francs, which to him was a very serious matter. Even reference to the fact that he was being pursued produced no response.

Even with this state of continuous and profound distraction, it could not be denied that he was much more irritable than before the onset of his illness. The author believes that the method he employed in obtaining the reaction time is preferable in this type of case to that of association, since the interpretation of the results gained by the latter is infinitely more complex and does not lend itself so readily to numerical evaluation.

WOLTMAN, Rochester, Minn.

POLIOMYELITIS. A STUDY OF THE 1916 PHILADELPHIA EPIDEMIC WITH A REPORT OF 717 CASES. T. H. WEISENBURG, Tr. Amer. Neurol. Assn., 1918.

Weisenburg has given the results of a thorough analysis and clinical study of 717 cases of poliomyelitis which came under his observation in Philadelphia during the epidemic of 1916. Of these, 388 were in males and 329 in females. The ages ranged between less than 6 months to over 30 years, 63 per cent. occurring between the ages of 6 months and 3 years. The total mortality was 22.8 per cent.

Preparalytic symptoms are considered under the heads of fever, respiratory and pulse rates, gastro-intestinal symptoms, nervous symptoms, respiratory symptoms, pain, retention of urine, sweating, chills, nosebleed and objective preparalytic symptoms. Objectively, the appearance in this stage was similar to that of meningitis, with stiffness of the head and neck, often with retraction of the head, general hyperesthesia, increased activity of the deep reflexes and positive Kernig sign. Fever was invariably present and of a very definite type. It was usually moderate, 100 to 103 degrees, with but slight diurnal variation. The duration was rarely over six or seven days unless some complication supervened. The respiratory and pulse rates paralleled the temperature curve. Nausea and vomiting were common; irritability and twitching occasional; delirium, stupor and convulsions rare.

The onset of the paralysis was noted quite uniformly on the second or third day, the paralysis rapidly extending to its most severe degree. Relapses were never observed. "A paralysis which tends toward increased destruction of function after improvement has been instituted should cause grave doubts as to the etiology being infantile paralysis." Pain in the head, neck and back was almost invariably present in the patients who were old enough to describe their sensations. The pain is not of neuritic, but of meningeal origin and disappears as the signs of meningeal irritation subside or when the intracranial pressure is relieved by lumbar puncture. The spinal fluid was found universally under increased pressure. The appearance was clear and colorless or with but faint opalescence. After standing, 10 per cent. of the fluids presented a small fibrin coagulum. A moderate lymphocytosis was found in the preparalytic stage and continued for about two weeks after the onset of the paralysis. An increase of globulin was found in 32 per cent. A colloidal gold reaction of the meningitic type was present in from 40 to 50 per cent. of the fluids during the acute stages. While "a clear or slightly opalescent fluid, poor in fibrin, reducing Fehling's solution and containing an increased number of cells chiefly of the mononuclear variety, are the most constant findings," there are no definite diagnostic features in the spinal fluid.

The extent of the paralysis could be determined a few days after the paralysis became manifest and was usually severe. The types of paralysis are divided into the following groups: (1) the spinal form; (2) the form resembling Landry's paralysis; (3) the pontine bulbar: (a) bulbar, (b) pontine, (c) pontine bulbar, (d) pontine spinal, (e) bulbar spinal, (f) pontine bulbar spinal; (4) encephalitic; (5) cerebellar; (6) meningitic; (7) abortive. In regard to the recession of paralysis, the cranial nerve palsies and manifestations of cerebellar involvement usually cleared up promptly. "Distal muscles of any particular extremity cleared up more rapidly than the proximal muscles. . . . The rate of improvement in any given extremity may be the same, but there is a more rapid improvement in the upper extremities when contrasted with the lower extremities."

Careful consideration of the question of contagion shows little evidence that the disease is personally contagious.

Various methods of treatment were used including urotropin by mouth, normal serum intraspinally and intravenously, mercuric chlorid solution intraspinally and arsenobenzol intravenously; also immune serum intraspinally and epinephrin intraspinally as advocated by Meltzer, but with little conviction as to the value of any of these agents.

This short, concise article is the result of a careful study of a large number of cases, and forms a valuable addition to our clinical knowledge of this disease.

Howe, New York.

WAR NEURASTHENIA, ACUTE AND CHRONIC. D. W. Carmalt Jones, F.R.C.P., Brain **42**:171 (Oct.) 1919.

In a careful study of 1,300 war neurasthenics, Jones emphasizes the physical aspects of the problem. Of the entire group, 612 cases were acute, in the sense that the neurosis was abruptly precipitated; 334 chronic, being usually the outgrowth of prolonged and insidious strain and in 354 the condition was complicated by organic disease. War neurasthenia is defined as: "A state of fatigue of the central nervous system, without organic change, manifested by asthenia, loss of physical and emotional control and disturbances of visceral functions." Briefly, the cause is a question of physical and emotional fatigue. As precipitating factors, personal "nerve" and resisting power are especially taken into account. The former concerns the soldier's record in civil life, information as to whether he has ever suffered from a "nervous" breakdown being of particular importance. As might be expected, a "bad nerve" record was fairly frequent. The author also found that the resisting power is less affected by the circumstances that apparently give rise to the neurosis, than it is by the personal qualities inherent in the individual.

Further, while this may be a considerable factor in a chronic case, it is of far less significance in an acute one. The symptomatic findings may be expressed in the following statement: "The commonest symptoms are those of common sensation in excess, disturbance of sleep, and disturbance of coordination; pure motor symptoms, with the exception of asthenia, are rare, visceral disturbances and also psychical symptoms are common though not invariable, and disturbances of the special senses are infrequent." Headache was present in 736 patients, disturbed sleep in 515, incoordination in 374, pain, other than headaches in 303, asthenia in 266, alimentary symptoms in 251, cardiovascular symptoms in 237, urinary symptoms in 204, psychic symptoms, aural symptoms in 191, respiratory symptoms in 104, ocular symptoms in 92, mutism in 85, paresthesia in 78, paresis in 41 and anesthesia in 37. The prognosis was usually good. Fifty per cent. of the patients with acute cases were returned to field duty and only 16 per cent. were sent to base hospitals. However, only 20 per cent. of the "chronic" neurasthenics were able to rejoin their former commands. The treatment was rest, graduated exercise, maintenance of discipline and mental occupation.

The author's recommendation that these patients be treated at a point far remote from the firing line must be seriously questioned. It is, of course, obvious that frequent artillery fire and bombing raids are not in themselves conducive to recovery from war neuroses. However, treatment near the front has certain advantages which outweigh its contraindications. The neurosis is less apt to become fixed when the patient is kept in the midst of stimulating military activity and the necessity of quickly returning to duty is constantly and strikingly suggested to him. In quiet areas, away from the "noise of guns," the urge is far less great and the stage of symptom-fixation is correspondingly easy (reviewer's opinion). I believe that the experience of divisional neuropsychiatrists who were with the A. E. F. will bear out these statements.

Strecker, Philadelphia.

A TRIPLE OBSERVATION OF FAMILIAL PSYCHOSES IN A FAMILY OF NEGROES. E. Terrien and R. Saquet, Progrès méd. **35**:72 (Feb. 14) 1920.

The authors report a negro family in which the father, a Senegal negro, and his two mulatto daughters, aged 18 and 19, developed psychoses. The former, a case of paranoia, was dominated by a paranoid idea, persistent during twenty years, on an invariable theme (his sonship to an Ethiopian king) without any intellectual enfeeblement. His original character shows inherent traits of a paranoid constitution—excessive egoism and pride. The daughters had dementia praecox with marked paranoid coloring. In them, intellectual impairment was progressive. The authors debate whether they were dealing with true familial mental disease as studied by Féré, Trénel, Fougue, Apert and Demay and conclude that they were.

These considerations are outlined: Demay has written that there is no parallelism between familial mental illness and familial affections of the central nervous system, such as Friedreich's ataxia, Landouzy-Déjerine myopathies or Thomsen's disease, because familial mental disease is not *sui generis*. Rather, the expression "familial psychosis" simply characterizes a resemblance which sometimes exists between psychoses in members of the same family. In the family cited, the paranoid feature is the common characteristic and alone justifies the term "familial" in their opinion.

Apert has pointed out that a psychosis to be classed familial must be outside of an exterior pathology (intoxication, infection, organic cerebral affections). Dumas does not believe that persuasion or example account for familial psychoses, though "persuasion may provoke the hallucinatory modifications." The authors consider that in their cases persuasion or example was not a factor as the death of the father occurred prior to the onset of symptoms in the daughter. They, perhaps, at this point, stress insufficiently the example set by the father in the preceding years—those during actual adolescence.

They elicit the law of progressive degeneration of Morel to explain one feature—namely, the dementia of the second generation in sharp contrast to the preservation of intellect in the father. They think that true paranoia cannot reproduce itself in a successive generation. Rather, it is a grain which begets in the following generation a thing more degenerative and destructive—paranoid dementia praecox.

Davis, New York.

A METHOD OF TREATMENT FOR NEUROSYPHILIS. J. A. Kolmer, J. A. M. A. **74**:794 (March 20) 1920.

Kolmer's technic seeks to avoid the disadvantage of the Swift-Ellis treatment by providing for the introduction of a larger amount of arsphenamin into the spinal canal. Advantage is taken of preliminary spinal puncture, which is said to increase vascularity of the meninges and favor absorption by the drug.

A "course" of treatment covers four weeks, and comprises four combined intravenous and subdural injections of arsphenamin at weekly intervals. The first injection is preceded by a week of daily mercurial inunctions and potassium iodid by mouth. The inunctions and the iodid are kept up throughout the course.

The technic of injection is:

Six-tenths gram of arsphenamin is dissolved in normal salt solution, neutralized and the volume brought up to 200 c.c.; 1 c.c. of this is removed for further use. The remainder is injected into a vein of the patient and immediately thereafter 25 c.c. of the blood are withdrawn from another vein. This is mixed with 4 c.c. of 10 per cent. sodium citrate solution and centrifuged. About 10 c.c. of the resulting blood plasma is then mixed with 0.1 c.c. of the arsphenamin solution (dosage 0.0003 gram). As high as 0.3 c.c. of arsphenamin solution may be used in subsequent treatment. The arsphenamized serum is heated at 56 C. for thirty minutes.

Spinal puncture is done at once with a small (1 mm.) needle to ensure a slow fall in pressure. About 30 c.c. are withdrawn and the serum then slowly injected by gravity or by syringe.

Blood withdrawn immediately after injection is said to contain more arsphenamin than that withdrawn one hour after injection. The total time of the operative procedure is about two hours. In case of reaction to arsphenamin the intraspinal injection may be reserved for twenty-four hours.

Haines, Philadelphia.

CEREBROSPINAL MENINGITIS. M. Aynaud, Progrès méd. **35**:45 (Jan. 31) 1920.

Regarding the four known types of meningococci, the author comments on the recent increased frequency of types B and C which now furnish more than one half of the cases of cerebrospinal meningitis. Type D remains rare clinically. Types A, B, C and D have the same cultural characteristics, including those of fermentation, and differ only according to their reactions to serums, where their agglutinations are specific. Therefore, the author insists that serum therapy should be based on type and that each case should receive the expert bacteriologic care necessary to establish the type before the serum therapy is begun. He has found only the most ultra aseptic technic satisfactory, and warns against any method other than drawing the spinal fluid into an autoclaved syringe which is then emptied into a flamed sterile tube.

The second portion of the article discusses the complication of pyocephaly and ventriculitis. In contrast to the fever, Kernig's sign and stiffness of the neck which are signs of meningeal infection, psychic, trophic and sphincteric troubles and violent headache are the symptoms of cerebral compression. They establish themselves as the ventriculitis develops. When the course indicates this complication, treatment should be by the ventricular route as well as by the lumbar. "If at the end of five or six days of correct and intensive treatment the fever and other symptoms persist along with continued purulence of the fluid, and if, in addition, a careful examination shows no septicemia or extrameningeal localization of the meningococci, I consider it proper to incriminate the ventricles and to have recourse to ventricular puncture." By the lumbar route, the author injects from 30 to 40 c.c. of serum after having withdrawn the same amount of fluid, but by the ventricular route he withdraws only from 15 to 20 c.c. and injects in serum an amount somewhat smaller. When ventriculitis has developed and the fluid remains purulent, he gives both a lumbar and ventricular treatment each day.

A third portion of the paper deals with anaphylactic sensibility, and procedures of desensitization are given. These are not new. In order to gain a perfect security against anaphylaxis, even though the patient is supposedly desensitized, he inaugurates intraspinal serum treatment as follows: He places 0.5 c.c. of the serum in a syringe and then aspirates 10 c.c. of the spinal fluid into it. This is allowed to mix and the mixture is immediately injected. Then with the needle remaining in place, he waits three or four minutes, after which he withdraws the customary 30 or 40 c.c. of fluid. After this, he finds the injection of the serum invariably free of untoward symptoms.

Davis, New York.

A PROPOS DU SONDAGE NASAL DES ALIENES. Will Boven (medécin à Cery), Schweiz. Arch. f. Neurol. u. Psychiat. **5**:99, 1919.

Boven lays stress on the importance of persuading patients to eat in a normal manner, and employs tube feeding only as a dernier ressort. As might be expected, patients, particularly those having mental disorders, react very differently, depending on the type of psychosis. Tube feeding by mouth usually is not only very difficult, but is also exceedingly uncomfortable, as far as the patient is concerned. Boven, therefore, uses the nasal route almost entirely. By experimenting on himself, and in response to inquiries among his patients, he concluded that the greatest pain in nasal feeding was the result of pressure on the inferior turbinate. This leads to pain referred to the superior molars, the nose and the temple of the homolateral side, the reflex path being the sphenopalatine ganglion and branches of the trifacial nerve. When a large tube is used, this is particularly severe. The pressure of a large tube in the vicinity of the larynx can also separate the vocal cords so that any regurgitated liquid readily enters the glottis. He has adopted the following procedure: The left nostril is sprayed with a 20 per cent. solution of cocain, after which a tube having a caliber of 5 to 8 mm. is inserted. At the moment the patient swallows, during which time the glottis is closed, the tube is rapidly passed into the esophagus and down to the stomach. Should the trachea have been entered, this is at once shown by a violent paroxysm of coughing which occurs after a few drops of water enter the lungs and through the inability of the patient to make an audible sound. In a selected number of mental cases he gradually discards the use of cocain and resorts to larger tubes, thus producing a gradual increase in the amount of pain, which not infrequently has the psychologic result of encouraging the patient to feed himself.

WOLTMAN, Rochester, Minn.

LES TROUBLES OCULAIRES AU COURS DE LA THROMBOSE JUGULAIRE DU CARDIAQUE. V. CORDIER and P. ROLLET, Progrès méd. **35**:108 (March 6) 1920.

A soldier with a history of rheumatic fever at 16 and again at 23 developed temporary cardiac symptoms at the age of 29. These responded to treatment but a year later (February, 1917) decompensation recurred and progressed to a fatal termination in four months. Ocular signs which are not easily explained even with the aid of careful necropsy search are the subject of the writers' interest.

The case was one of mitral stenosis and insufficiency with pericarditis. A thrombophlebitis of the great veins draining the right upper extremity developed and there were multiple and repeated infarctions of the lungs. For forty-eight hours before the patient died there was unilateral exophthalmus (right) with diplopia and also abolition of the light reflex in the right eye. The diplopia was not accompanied by ocular paralyses nor the light reflex loss by loss of the reflex on accommodation. There was no pain on pressure on the eyeballs, no signs of meningitis, no vertigo, no troubles of ideation or of language, and audition was normal. The eyelids were not edematous and showed no painful or indurated bands. The orbital and peri-orbital regions were free from swelling. The conjunctivae showed no injections. There was slight congestion of the fundus though the large vessels were not turgescent and there was no retinitis or hemorrhage. Necropsy showed a thrombophlebitis of the right innominate vein beginning at the superior vena cava. It extended upward 3 cm. in the internal jugular and 6 cm. in the external jugular and extended laterally a distance of 2 cm. in the subclavian. Careful search was made in the cranium. There was a slight degree of meningeal congestion. There were no retro-ocular effusion, no tumor, abscess or aneurysm. The arteries were normal and nothing abnormal was found in the veins or sinuses. The cavernous sinus was especially explored and found clear as were the tributaries, the ophthalmic veins. All the bony sinuses were free of infection.

The few ocular signs suggesting a cavernous sinus thrombosis but lacking several features of that syndrome are not readily explained, in the writers' opinion. In the absence of all other causes, however, they were forced to conclude that it resulted from the venous stasis occasioned by the thrombosis centering in the innominate. They mention that this case appears not to accord with the experimental conclusions of Ferrari, working on dogs.

Davis, New York.

VISION IN OCCUPATIONAL NYSTAGMUS. L. Weekers, Am. J. Ophthal. **3**:162, 1920.

The author draws an analogy between hemeralopia and nystagmus as observed among the soldiers during the recent war, attributing these conditions to muscular and nervous fatigue, and he applies this principle to occupational nystagmus.

He believes forced marches, night work, excessive physical fatigue and the nervous strain of bombardment prepared the field for these conditions, in a similar manner as the poor illumination, unhygienic surroundings of the mine and fatigue do for miners' nystagmus.

He quotes Professor Nuel who insists that two factors destroy the muscular equilibrium usually controlling vision; namely, the failure of the fovea to function and the loss of binocular vision. He explains the first factor by recalling that in dim light the retina sees with the rods and not the cones, and as there are no rods in the fovea it is necessary for the eye to shift its focus 15 to 20 degrees from the fovea for vision. This groping tends to cause the nystagmus of miners, as the lighting is poor and the unhygienic surroundings quickly leads to fatigue. The second factor, the loss of binocular vision, he explains by the fact that the coal has angular and crystalline facets and the lamp lighting these facets is reflected, thus each eye sees different facets, destroying binocular vision, and easily fatiging the eye.

The author believes that miners' nystagmus does not entirely consist of ocular displacements, but includes insufficiency of retinal adaptation, and that dark adaptation is a function not only of the retina, but also of the cerebral centers and as such may be especially affected by over fatigue.

Scarlett, Philadelphia.

HYPERGLYCEMIA IN MENTAL DISORDERS. F. H. Kooy, Brain **42**:215 (Oct.) 1919.

This paper presents the results of an extended series of observations of the blood sugar of various types of psychotic persons made before and at short intervals after the ingestion of a standard breakfast. The values obtained from twenty normal persons following the same schedule are used as the basis of comparison. In ten cases of dementia praecox, nine cases of dementia paralytica and eight cases of epilepsy no valid differences from normal persons were observed either in the absolute amounts or in the course of the blood sugar curve after eating. In melancholia, however, there appears to be a tendency not only for the initial values to be higher, but also for the blood sugar increase after eating to be greater than in normal persons, nineteen cases being reported. The same phenomenon of higher blood sugar after eating was observed in four cases of neurasthenia or psychasthenia and in six cases of amentia (confusional insanity). The results obtained from five cases of mania were variable. The author attributes the production of the so-called hyperglycemia to the relatively greater emotional reactions occurring in these types, since the patients showing the greatest degree of anxiety had at the same time the highest amounts of sugar in the blood after eating. He correlates this with an hypersecretion of epinephrin, or better an overexcitement of the sympathetic nervous system, clinical evidence of which is given by the higher blood pressures, the pupillary dilatations, the diminishing reactions, and the reduced intestinal activities obtaining in these patients.

Hammett, Philadelphia.

THE "NERVOUSNESS" OF THE JEW. A. Myerson, Ment. Hyg. **4**:65 (Jan) 1920.

Myerson's masterful presentation of his subject should not pass unnoticed. His reasons for the predilection of the Jew for the psychoneuroses are interesting and convincing. The predisposition springs not from biologic but from "social heredity" which means the influence of groups of life factors handed down for generations in the growth and development of a race. Marked religious differences have given the Jew the stamp of unlikeness and therefore, he has been prejudged as different and unsociable. Further, "he was excluded from all occupations, in the pursuit of which the manual-motor side of his nature might find expression." This conditioned a dislike for the occupations from which he was shut out and gave him an inferior physique. By force of circumstances he developed largely into an urban, sedentary, cerebral type. Living in constant dread of ridicule, degradation and massacre, a lowered threshold for fear stimuli was acquired. The effect of this emotion on the material organism must also be considered. In this connection it is often stated that the Jew is particularly liable to arteriosclerosis and diabetes. In favor of social as against biologic heredity, the author cites the rapid amalgamation of the Jew with his Gentile fellows when conditions are favorable and the character and habit modifications that this amalgamation effects.

Strecker, Philadelphia.

THE LIMITATIONS OF TINEL'S SIGN IN PERIPHERAL NERVE INJURIES. Byron Stookey, Neurol. Bull. **2**:380 (Oct.) 1919.

Stookey discusses both the importance and the limitations of Tinel's sign, signe du fourmillement, which is obtained by pressing on or tapping the distal stump of a nerve trunk containing regenerating axons, and describes three cases to illustrate various points. The sign in Stookey's opinion has only qualitative value and is very important when taken in conjunction with other signs of regeneration but furnishes little guidance for operation when taken by itself, as its presence does not preclude the existence of complete interruption. The sign, according to Stookey, is found (1) in cases where continuity exists and the neuraxes can penetrate into the distal stump, (2) when despite more or less complete interruption a sufficient number of neuraxes penetrate the distal stump, but the interruption is sufficient to prevent ultimate functional return, and (3) when the interruption is absolute but a few stragling neuraxes, either from the proximal stump or some of its adjacent branches, find their way by a devious path into the distal stump. The sign, therefore, even though it be present and progressive should not be considered an evidence of satisfactory regeneration unless, when the anatomic field is known, confirmatory evidence is also found. In so far as it indicates qualitatively a downgrowth of neuraxes, it is a valuable sign; when it is progressive and is associated with a recession of the field of sensory loss, etc., it is indicative of regeneration. Alone it is not capable of quantitative determination and is devoid of definite significance.

Wechsler, New York.

EPIDEMIC ENCEPHALITIS AND KATATONIC SYMPTOMS. Earl D. Bond, Am. J. Insan. **76**:281 (Jan.) 1920.

The author emphasizes the katatonic aspects of five interesting cases. Three were undoubted examples of epidemic encephalitis; the fourth was an acute hemorrhagic encephalitis, and the fifth a katatonic episode in the course of a chronic depression. Bond's paper is a distinct addition to the literature of katatonia and his observations are added proof in favor of its organic nature. The evidence cited should make one realize the futility of attempting to find a purely psychogenic origin for this symptom. That its occurrence, in some instances at least, may be a matter of fleeting but none the less actual brain involvement, seems to be a reasonable and plausible hypothesis.

Attention is called to our vocabulary deficiency in the description of tonal muscle abnormalities. Much valuable clinical evidence must be lost because we cannot or rather do not suitably reproduce on paper the particular variation of the symptom observed at the time of examination.

Strecker, Philadelphia.

Society Transactions

CHICAGO NEUROLOGICAL AND THE CHICAGO SURGICAL SOCIETIES

Joint Meeting, Jan. 2, 1920

Charles E. Kahlke, M.D., President of the Chicago Surgical Society, *in the Chair*

SURGICAL ASPECTS OF PERIPHERAL NERVE INJURIES. Presented by Dr. Dean Lewis.

I prefer to demonstrate this evening some of the results that may be obtained in peripheral nerve surgery, rather than to discuss formally the many problems presented in injuries of this type.

Case 1. — This patient was wounded Oct. 27, 1918. He sustained four wounds. The internal condyle of the left femur was fractured and subsequently removed. As you see, there is a large scar over the left shoulder posteriorly and also one over the sacrum. The fourth wound is directly over the course of the lower part of the right musculospiral nerve which was evidently divided, for paralysis was noted immediately. On Feb. 21, 1919, the musculospiral nerve was exposed in the groove between the brachialis anticus and the supinator longus. The nerve had been completely divided. The neuroma on the end of the proximal segment was resected until healthy neurofibrillae were exposed and the scar tissue of the end of the distal segment was also removed. Two tension sutures of fine catgut were inserted and the epineurium was then closed with fine chronic catgut.

Definite evidences of return of motion were noted about five and one-half months after suture. As you can see, there is now almost complete return of power in all the muscles supplied by the musculospiral nerve. The extensors of the fingers react slowly. The patient states that he cannot throw a ball as he cannot release the fingers rapidly. Marked hyperesthesia of the skin supplied by the musculospiral nerve was noted during recovery. This has gradually subsided.

Case 2.—This boy was wounded during the first week of November, 1918. The right musculospiral nerve was divided by a machine-gun bullet which perforated the arm, apparently passing through the humerus without fracturing it. The nerve was sutured on March 17, 1919. He has complete return of motor function in the radial extensors and also some in the common extensors of the fingers. There is no evidence of return of function in the abductor of the thumb. Regeneration has occurred through a neuroma. At the point of suture there is a definite fusiform enlargement of the musculospiral nerve which is painful on pressure. Regeneration has occurred rapidly, however, as evidences of return of motion were noted five and a half months after suture.

Case 3.—This boy was wounded in the Argonne Forest. The musculospiral nerve was divided on the inner side of the arm just before it entered the groove. It is somewhat difficult to suture the nerve in this position, as it lies somewhat deeply and bleeding from the superior profunda artery may be troublesome. The operation was performed on April 22, 1919. Motor function is present in the radial extensors. The power is good. There is some evidence now of return of motion in the extensor communis digitorum. The abductor of the thumb is still paralyzed.

Case 4.—This patient presents some interesting features. He was wounded in the Argonne Forest, sustaining a gunshot fracture of the humerus near the middle and an injury of the musculospiral nerve. The wounds were completely healed and a roentgen-ray examination of the humerus was, therefore, not made. When the incision to expose the divided nerve was made, a sequestrum surrounded by granulation tissue and some pus was found in a cavity directly beneath the divided nerve. The sequestrum was removed, the granulation tissue curetted out and the cavity iodized. An end-to-end suture of the divided nerve was performed and the wound closed. Much to my surprise the wound healed by primary union. Up to ten days ago there has been absolutely no evidence of return of function. Suddenly the patient noted that he could extend his wrist. Since then the improvement has been rapid. Sudden return of motion, as in this case, has been noted in other cases that we have observed.

Case 5.—This patient had a combined median and ulnar lesion. Both nerves were divided in the middle of the arm. You can see the large scar that followed the healing of a wound caused by a piece of high explosive. At the time he was operated on there was a distinct lymphedema of the skin below this partially constricting scar. At the time of operation conditions seemed unfavorable because of the lymphedema and the difficulty of making an end-to-end anastomosis. The suture was made under some tension. I believe that the results are surprisingly good, for the patient has regained almost complete use of the muscles supplied by the median nerve and there is some return of motion in those supplied by the ulnar. One of the most striking changes is the appearance of the hand; the color is better and there has been a decided improvement in the general appearance.

Case 6.—This patient was wounded on September 22. He received a high explosive wound of the left foot and one of the posterior aspect of the left thigh. About 3½ inches of the sciatic nerve were destroyed. He was operated on March 19, 1919. An end-to-end suture was performed after the segments of the sciatic nerve were mobilized and the knee flexed to a right angle. About one and a half months ago power returned in the muscles supplied by the internal popliteal nerve. The muscles supplied by the external popliteal are still paralyzed. There has been decided improvement in the appearance of the leg and foot. Both appear larger and are more solid. The atrophy is much less marked.

The next two cases are instances of recovery or improvement after neurolysis. The first patient received a high explosive wound. The left humerus was fractured and the left musculospiral nerve injured. You can see that there is considerable deformity. According to the clinical findings, the musculospiral nerve was completely divided. On April 26, 1919, the musculospiral nerve was exposed. When dissected out of the scar, it had a frayed appearance, but four funiculi were found passing from the proximal to the distal segment. This patient has now extension of the wrist and beginning extension of the fingers. There is, however, no return of power in the abductor longus pollicis.

The second patient was wounded October 5. According to the symptoms, there should have been found a complete division of the sciatic. When the nerve was exposed on July 30, 1919, it was found embedded in scar tissue, but there was no anatomic division. The nerve was dissected out of the scar tissue and a muscle neurolysis performed. The internal popliteal part of the great sciatic presents evident signs of recovery, but there is no improvement in the external popliteal. It might be argued that in these two cases recovery would have occurred if no operation had been performed, but I do not believe that any one can deny that there is a very definite relationship between the operation and recovery.

The cases presented demonstrate that the musculospiral nerve recovers rapidly after suture. The median, ulnar and internal popliteal also recover fairly rapidly. The external popliteal recovers slowly. After sciatic suture, return of function of the muscles supplied by the internal popliteal is noted much earlier than of those supplied by the external popliteal. This is somewhat surprising as the external popliteal is usually easily exposed and the suture easily made. The large amount of muscle that has to be reinnervated might account for the slow return, but the same amount or more has to be reinnervated after suture of the internal popliteal nerve.

End-to-end suture should be performed when possible, for it is the only procedure that gives a high percentage of successes. Mobilization of the segments, displacement of nerves and proper posture will often enable one to make an end-to-end suture, even when at first it seems impossible. The suture material apparently makes no difference. We have employed epineural sutures of fine silk and in other cases have employed transfixion sutures of fine chromic catgut. It is essential to secure as accurate apposition as possible of the nerve ends and to close the epineurium carefully, so that the neurofibrillae will be prevented from straying into the surrounding connective tissue.

TRANSPLANTATION OF PERIPHERAL NERVES. By Dr. G. Carl Huber, Ann Arbor, Mich.

Owing to a clerical error, an abstract of Dr. Huber's remarks appeared in the abstract department, page 437, of the April issue of the Archives.

CLINICAL SIGNS OF PERIPHERAL NERVE INJURY AND REGENERATION. Presented by Dr. Lewis J. Pollock.

Dr. Pollock described such signs of nerve injury and regeneration as appeared to him to be most important from the study of cases coming under his direct observation.

He called attention to the influence of supplementary movements on the clinical picture of paralysis (Arch. Neurol. & Psychiat. **2**:518 [Nov.] 1919). He then described the influence of nervelap on the relatively early return of sensation to pin-prick (Arch. Neurol. & Psychiat. **2**:667 [Nov.] 1919). He was unable from his experience to differentiate anatomic from physiologic interruption, and stated that in general the course of the clinical picture is much more important than any group of signs for the purpose of determining the severity of a lesion. Among the signs of a severe lesion he found that analgesia to severe pinching occurred in that area insensitive to pin-prick occupying the isolated sensory supply of the nerve. He stated that absence of demonstrable atrophy some months after injury is not an indication of a reparable injury. Absence of pain when the trunk of the nerve is subjected to pressure below the seat of the lesion was demonstrable in many severe lesions. On the other hand, not a few irrecoverable lesions showed the preservation of pain to such pressure. Absence of pain to pressure on the paralyzed muscles was likewise found to be an unreliable sign. Trophic changes could be employed as an indication of the severity of the lesion only when judged in the light of the presence of other signs. This was particularly true of ulceration which occurred only in analgesic areas, cessation of growth of nails in immobilized extremities and atrophy of bone under similar conditions.

Many cases of complete so-called physiologic interruption of a nerve showed their first sign of regeneration at such a time as one would expect it to occur, had the nerve been divided at the time of injury and sutured. It is reasonable to assume that in this type of severe lesion complete descending degeneration had occurred and conditions permitted the subsequent regeneration of the axon. Return of pain on pinching of the skin was frequently the first sign of nerve regeneration. Spontaneous aching, and more frequently a sensation of a "different feeling," were found. Tinel's sign had not been found to be of any precise value.

Relative to the return of sensation, only when that portion of the skin representing the isolated supply of a nerve becomes sensitive can it be said that regeneration is present. Under this condition at no time did protopathic sensibility return before epicritic. Arrest of atrophy and return of tonicity in those patients recovering some months after injury are not valuable as signs of regeneration. Following resection and suture, reaction to faradism did not return before motion. In patients recovering a number of months after the injury, electrical reactions were not of great value in the determination of regeneration. In the recovery consequent to resection and suture, the various peripheral nerves showed a distinctive picture in the relatively constant progression of recovery of the several muscles supplied by them.

DISCUSSION OF REPAIR OF PERIPHERAL NERVE INJURIES

Dr. Ernest Sachs, St. Louis: I do not feel that I can add to this subject. In addition to hearing the papers this evening, I also had the good fortune this afternoon to see Dr. Huber's beautiful specimens. They added tremendously to the interest of what I have heard tonight, and that has cleared the atmosphere remarkably on the subject of nerve surgery. After a study of this large amount of material, both clinical and experimental, certain things long thought and taught about peripheral nerve surgery, can be discarded for all time. The various old methods of trying to bridge defects by making flaps, and so on, have now been shown to be unsatisfactory. With proper methods peripheral surgery and peripheral regeneration are perfectly feasible.

Many of you may remember—I think it was three years ago—that at the meeting of the Clinical Congress in Chicago we heard some pessimistic remarks from Sir Berkeley Moynihan on the subject of peripheral nerve surgery. Undoubtedly these pessimistic reports were due to the fact that the fundamental principles of nerve suture as now known were not followed at that time: first of all, exposing the fibrillae in the proximal and peripheral stumps; second, a dry field; third, doing whenever possible, primary suture or direct suture and closing the neurilemma sheath so that connective tissue will not grow in.

One other point that was not emphasized, but which should be covered, is that the handling of nerves is very different from the handling of other tissues of the body. A nerve must be handled with great gentleness. Another thing which my experience has shown to be very important is to keep the nerve warm, and not allow it to become chilled.

The only phase of the work on which I have had any practical experience confirms absolutely what Dr. Huber said. It was first described by Rehn who emphasized its great value in applying it over defects in the brain. About eight years ago I tried that out experimentally and became convinced that his observations were absolutely wrong; that the fat was completely replaced in a short time by a dense mass of connective tissue, and several cases to which I applied this clinically, I had an opportunity later on of again opening and found the same thing occurring. The fat is replaced by a dense mass of connective tissue.

One point that Dr. Pollock brought out; namely, that it is impossible to tell whether a nerve is anatomically or physiologically divided, seems to me a valuable contribution. What he said about the length of time after which a nerve may still regenerate, I do not question, but his statement might lead to faulty surgical treatment. If it is possible for a nerve to regenerate seven or eight months after an injury, I do not believe it is advisable to wait as long as that to determine whether or not the nerve is absolutely divided. If Dr. Pollock believes that, I must take issue with him. If there is evidence that the nerve is completely blocked, I believe the proper procedure is to explore the region, determine the nature of the block and, if necessary, take steps to correct it.

I do not wish to discuss his statements concerning the time of recovery of a nerve because he has had much material to work on, but anyone who has busied himself for a long time with neurology cannot help feel a little disconsolate, as we all do, when something we have banked on for many years has been undermined; in other words, certain props have suddenly been knocked from under one. I refer particularly to what he said about the work of Head in regard to peripheral nerve disturbance. His evidence is extremely interesting; it is difficult to contradict; but he has not absolutely refuted the work of Head and his various co-workers which extends over many years. I do not quite see how he explains the return of sensation in the ulnar anesthetic area by the overlap, for it seems to me that if the overlap controls a large part of that area, the sensation of that area—that overlap—ought to be present immediately after the injury. In other words, the only anesthetic area should be the exclusive area supplied by the ulnar nerve. As I understand his observations, and I have read his article on the subject, there is a complete loss of sensation, and after a short time some sensation returns in that area. I cannot quite see why there should not be only the exclusive area supplied by the ulnar nerve as he has worked it out, and from the beginning. I hope Dr. Pollock will answer that point in his closing remarks.

Dr. G. Carl Huber (closing, on his part): I have enjoyed listening to the presentation of the neurologic side of this subject by Dr. Pollock, and was personally very glad to hear the questions asked with reference to interpretation of facts presented, because there has not been unanimity of view on questions under discussion. Referring to Dr. Pollock's work, in the first place, there are no hard and fast boundaries of peripheral nerve distribution. I think the anatomists will have to change their figures—at least for so-called exclusive areas of peripheral sensory nerve distribution. I think Dr. Pollock has helped us a great deal in that direction, and I can readily see that there may be an apparent overlap in sensory areas, more than is generally recognized. The sensory nerve supply for a particular area, for instance, the peripheral ulnar field, need not be exclusively supplied by the ulnar; other bordering nerves may be educated in the course of a few weeks to give perception from the borders of the same area. I am confident that it is not necessary in practical work to lay undue stress on Stoffle's studies on the specific funicular structure of nerves. Any one who has worked experimentally with regeneration of nerves realizes that from ten to fifty new neuraxes may bud toward the periphery from a single central neuraxis, and fifteen or twenty new neuraxes are often found centrally in a single old neurilemma sheath. No matter how carefully primary suture is made, there is a great tangle of these new nerve fibers as they pass through the connective tissue of the wound, and especially is that the case with secondary sutures. A large number of fibers pass from the stump along the transplant in the connective tissue surrounding the transplant. I am sure sensory nerve fiber branches reach the motor nerves, and that central motor nerves reach the distal sensory nerves and are maintained for a time. They make no distal connection and in time degenerate. I am confident there is never complete regeneration of the peripheral stump, and to some extent there needs to be reeducation of the nerve centers after every regeneration of the peripheral stump. The anatomic findings often give very distinct motor and sensory recovery without full functional return.

Dr. Pollock (closing): I did not wish to be understood as saying that no operative procedure should be undertaken in the case of a severely injured peripheral nerve in less than eight or nine months: The point I wished to emphasize was, that if such an injured nerve can at times recover spontaneously, if any surgical interference be attempted it should not be in the nature of resection and suture.

I cannot thoroughly discuss the question of nerve overlap at this time. However, I might point out several facts. First, while the work of Head and his co-workers has been accepted largely by physiologists and neurologists, not all recent publications of physiologists confirm their findings. We need only refer to the work of Boring which proves definitely the educability of overlap nerves. If three nerves supply the entire leg below the knee and we cut two of them, the third is left. If, therefore, we have a lesion of the internal saphenous and the external popliteal the sensibility present is subserved by the internal popliteal. If we have a combined lesion of the internal saphenous and internal popliteal the external popliteal is left. By using this method of residual sensibility we can reconstruct the distribution of the total sensory supply of the various peripheral nerves. I maintain that the early return of sensibility to prick pain occurs only in such areas as are within the areas of nerve overlap of adjacent nerves. These areas constitute that part of the residual sensibility of a peripheral nerve which extends beyond the limit of the anatomic sensory distribution of an adjacent nerve. If this early return of pain sense always appears in areas of nerve overlap; if it never appears in such an area when the nerve supplying this area with sensation is cut; if, when it has appeared and the adjacent nerve is subsequently severed, it disappears, and, finally, if it has appeared and resection and suture of the originally injured nerve have no effect on this sensibility, it seems to me that my point that this early return of sensibility to prick pain is due not to nerve regeneration but to nerve overlap, has been proved.

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, Feb. 19, 1920

MYOTONIA ATROPHICA. Case presentation by Dr. Ralph C. Hamill.

The patient was a man who gave a history of difficulty in relaxing his grip. In military service he did not handle his gun well. One brother and one sister were normal. The muscles were good with the exception of the shoulder girdles and forearms. The trapezius was rather prominent above the scapular outlines, which were abnormally prominent because of the small size of the supraspinatus. The extreme atrophy of the sternocleido mastoids also emphasized the prominence of the trapezius. The upper deltoids were somewhat wasted; there was increased prominence of the lower portion of the vertebral border of the left scapula; the pectorals were quite good. The flexor muscles of the forearms were wasted, especially the left, the patient being left-handed. Flexion and extension of the wrists were weak, especially the left. The grip was poor, mostly exerted between the thumb and last phalanges of the fingers. Flexion of the hand was poor on the left, fair on the right. The thumb muscles were involved. There was no marked atrophy in the hands. Pronation and supination were weak; abduction and adduction of the fingers was fairly good; he could not make a fist. Flexion of all phalanges was nil or nearly so. Apposition of the thumb was weak. There were no atrophies in the lower extremities.

Reflexes: Plantar reflexes were normal; the right ankle jerk was absent; the left was present only with reinforcement. The right knee jerk was diminished, the left less so. Abdominal reflexes were normal, wrist jerks were absent, the left triceps was present, the right was absent except with Jendrassik. The jaw jerk was absent. The eyelids did not close completely. Movements of the forehead were very slow; those of the lips of small amplitude; he could open his mouth well and the tongue protruded without tremor. After biting hard he had difficulty in opening his mouth, but after once opening it there was no further difficulty. Strength of the masseters was normal, as was also movement of the eyes. Sense of passive motion was slightly diminished in the toes; normal in the fingers. The sternocleidomastoid muscles were almost absent. He could hop well on either foot. There was complete absence of fibrillary or other twitching. After gripping firmly he had difficulty in relaxing or in extending the fingers, but after exercising a few times he could relax them more easily. As bearing on the possible glandular origin of his trouble, it may be noted that the testes were undescended.

DISCUSSION

Dr. G. B. Hassin asked whether any myotonic reactions were present.

Dr. Hamill replied that he could not determine any myotonic electric reaction. The case was interesting because the disease was unrecognized in the Army and the man was accounted a serviceable soldier.

FURTHER OBSERVATIONS ON THE PRESENCE OF ARSENIC IN THE SPINAL FLUID. Dr. George W. Hall.

This paper is published in full in this issue.

DISCUSSION

Dr. H. I. Davis asked whether the experiments had influenced Dr. Hall's decision as to the use of neoarsphenamin.

Dr. Julius Grinker asked whether Dr. Hall had made comparisons in the amount of arsenic found in the fluid following the intravenous as compared with the amount found following the intraspinal injections.

Dr. Ralph C. Hamill asked whether Dr. Hall had considered leukocytosis as evidence of inflammation of the membranes, and whether the inflammation was not itself curative.

Dr. A. B. Yudelson asked whether Dr. Hall had noted the particular type of cerebrospinal syphilis in which these fibrins were observed.

Dr. Hall, (closing) in reply to Dr. Grinker, said that he had tested the spinal fluid for arsenic several times after intraspinal injections and had concluded that it was a dangerous and inefficacious procedure. He did not feel able to answer Dr. Hamill's question.

As to whether the results were just as good from injecting the autoserum as those of the Swift-Ellis method, he had no definite data, but he believed that the clinical results were equally good. The reactions were very different in different persons when the autoserum was used. Such marked differences of reaction were not observed in the use of intraspinous mercury. If the results depended entirely on irritation of the meninges he was inclined to think that the mercuric chlorid or mercuric succinimid produced a more uniform reaction, although more painful. One one-hundredth grain of mercuric succinimid or mercuric chlorid could be safely used, but $\frac{1}{50}$ or $\frac{1}{25}$ grain was hazardous.

BRAIN ABSCESS WITH PRESENTATION OF GROSS SPECIMENS.

Dr. Peter Bassoe.

In Dr. Bassoe's absence, Dr. Hassin presented specimens and related the histories of the following cases:

1. *Acute Traumatic Brain Abscess.*—A child, 17 months old, received a superficial wound 1 cm. to the right of the median line at the vertex. A week later pus appeared and two weeks after the accident the child was admitted to the hospital with no symptoms except discharge from the wound. Roentgen-ray examination revealed a small circular perforation of the skull at the site of injury. Fever gradually developed and three weeks after the injury the wound was explored. The dura protruded through the perforation of the skull. On aspiration pus was obtained and drainage established. The child died two months later and the abscess was found to extend almost to the occipital pole posteriorly and a short distance into the frontal lobe anteriorly.

2. *Chronic Frontal Lobe Abscess Following Gunshot Wound.*—A man, 21 years old, was admitted to the hospital in a semicomatose condition, moaning with pain in the head. He had bilateral choked disks, slight fever, leukocytosis of 14,900 and a discharging sinus in the right frontal region. It was learned that he had been in good health for years until the morning of the day of admission, also that eight years previously he had sustained a gunshot wound of the right side of the forehead. He died twelve hours after admission and the necropsy revealed a large thick-walled abscess which occupied the anterior half of the right hemisphere. The dura was adherent both anteriorly

and externally, and beneath the latter point of adhesion a flattened piece of bullet was detected. Apparently the patient had for eight years harbored an abscess and the bullet fragment without discomfort.

3. *Fracture of Left Temporal Bone with Abscess at the Site of Contrecoup in the Right Frontal Lobe.*—A man, 29 years old, who had been struck by an automobile and sustained a fracture of the left temporal bone with hemorrhage from the nose and ears, developed mental confusion, slight aphasia and weakness of the whole right side, including the lower part of the face. Naturally, a left-sided brain lesion was expected, but on operation six weeks after injury, nothing was found except a small accumulation of clear fluid. The patient improved, but three months later developed purulent meningitis and died. The brain, when obtained by Dr. Bassoe, was in bad condition, but it was possible to determine that in addition to recent meningitis and superficial necrosis at the site of operation, there was a small abscess near the under surface of the right frontal lobe surrounded by necrotic brain substance and the overlying pia showed rusty discoloration. There was also a rust colored softened area on the under surface of the left frontal lobe, but this had not suppurated. It was thought that the hemorrhage and necrosis at the site of contrecoup in the frontal lobe were the direct result of the injury and that infection from the blood stream had taken place later. No focal symptoms were attributable to this lesion which involved a silent area. On the other hand, the rather slight lesion of the left hemisphere involving a highly important functional area, caused distinct focal symptoms.

4. *Brain Abscess Secondary to Lung Abscess Following Tonsillectomy.*—A woman, aged 36, had a lung abscess drained three weeks after tonsillectomy under ether. Eleven months later she developed severe headache, vomiting and optic neuritis. The spinal fluid gave a negative Wassermann test, a cell count of 957, a positive globulin and Lange gold test. Necropsy revealed an abscess cavity which occupied the right frontal and temporal lobes.

5. *Cerebellar Abscess with Abscess of Sphenoid Sinus.*—There were the postmortem findings in a girl 13 years old, in whom a diagnosis of brain tumor of undetermined location had been made. There had been slight fever and a leukocyte count of 10,400 prior to a subtemporal decompression, following which there was relief for two days and then increased papillitis, fever and leukocytosis of 16,400. No evidence of sinus disease had been found, either by roentgen-ray examination or other methods. The patient died about six weeks after the onset and two weeks after the operation. There was a walnut-sized abscess in the left lobe of the cerebellum, and the right sphenoid and posterior ethmoid sinuses contained a large quantity of mucopus, which was not under any tension.

(These cases will be found reported in full in the May number of the *Medical Clinics of North America.*)

DISCUSSION

Dr. Julius Grinker said the mortality from brain abscess was high because it was difficult to localize and difficult to drain; because there is a tendency for abscesses to multiply, or for hour-glass abscesses to form. Drainage should be maintained until the area was completely drained. He thought it was the multiple abscesses that killed. In one instance a frontal lobe abscess was diagnosed, operated on and the patient made a clinical recovery; but something was still wrong. The choked disk, previously well marked, had cleared up for only a short time; indeed, it became more intense than before operation. Undoubtedly this meant that pressure was still present. When the symptoms became more aggravated the patient was again operated on but the surgeons missed the other abscess, found postmortem.

CONTRIBUTIONS TO THE STUDY OF CRUVEILHIER'S "TUMEURS PERLEES." DR. PERCIVAL BAILEY.

Since Cruveilhier, in 1829, described peculiar pearly tumors at the base of the brain, about sixty-two cases have been reported. They occur as about 0.2 per cent. of brain tumors. "Cholesteatoma" is the term usually applied to these growths, which is unfortunate, because often they do not contain cholesterin and moreover congenital cysts, collections in the middle ear after chronic otitis media, etc., contain cholesterin crystals but have nothing in common with these pearly growths.

The favorite location is on the base of the brain, beneath the dura but outside the brain tissue proper. They are occasionally found in the ventricles. Seven cases are described in the fourth ventricle. Grossly, they startlingly resemble mother-of-pearl. The outer portion is distinctly laminated. The interior looks like dry cottage cheese and easily crumbles. The surface peels away from the surroundings.

The microscope reveals four distinct layers which may be termed from without inward, stratum durum, stratum granulosum, stratum fibrosum and stratum cellulosum. The stratum durum consists of an almost homogeneous substance with occasional pyknotic nuclei. The stratum granulosum is made up of flattened cells containing definite nuclei, and granules which stain well with cresyllightviolet, evidently of keratohyalin. The stratum fibrosum consists of layer after layer of homogeneous material containing no evidence of cellular structure. The stratum cellulosum is composed of polygonal cells with heavy cell walls showing only occasionally a poorly preserved nucleus. The interior of the mass is a mixture of broken-down cells and fatty material. Cholesterin crystals are said to be occasionally found.

There are many theories of their origin but the structure is so definitely epithelial that their origin from ectodermal inclusion is generally accepted. They have hitherto been of more interest to the pathologist than to the surgeon. So far as I know, the cases here reported are the first to be successfully operated on.

CASE 1.—Peter Bent Brigham Hospital; surgical number 10302; housewife, aged 28.

Complaint: Headache, vomiting, failing vision, tinnitus and unsteadiness in walking.

Chronology of Symptoms: For five years headache and vomiting. For one year pain in the upper teeth on the right side. For eleven months failing vision and tinnitus. For six months staggering gait. For several weeks weakness and tremor of the left hand and emotional instability.

Positive Neurological Findings: Nasal margin of left optic disk definitely obscured by edema. Slight and poorly sustained nystagmus on looking to right and left. Auditory acuity diminished in left ear. Slight ataxia and adiadokocinesis of left hand on long exertion. Epigastric and abdominal reflexes absent. Gait staggering with perhaps deviation to left. Slight ataxia of left leg. Emotional instability. Caloric tests elicited, slight reactions from the right ear and marked reactions from the left.

Diagnosis: Intracranial tumor involving the cerebellum.

Operation: Suboccipital exploration by Dr. Harvey Cushing. Beautiful pearly tumor exposed completely filling the fourth ventricle, flattening the cerebellum and extending down into the spinal canal. The tumor was completely removed, leaving a smooth, glistening, dilated fourth ventricle.

Postoperative Progress: Patient developed a sixth nerve palsy which rapidly cleared up, and she was discharged a month after operation. When last seen eight months later, she had gained 20 pounds in weight and there were absolutely no neurologic findings.

Case 2.—Peter Bent Brigham Hospital; surgical number 11152; schoolboy, aged 13.

Complaint: Failing vision.

Chronology of Symptoms: For six years severe frontal headaches which lately had disappeared. For eleven months gradual failure of vision in right eye which progressed to total blindness in six months. For six months gradual failure of vision in the left eye.

Treatment Before Admission: Right subtemporal decompression about a year before admission.

Positive Neurologic Findings: Well nourished but not fat, 6.64 kilogram below standard weight for his age and 7.7 cm. below standard height. Hands small but not typically pituitary. Genitalia showed evidence of early adolescence. No signs of dystrophy. Blindness in right eye and temporal hemianopsia in left. V. A. left eye 10/70. Bilateral primary optic atrophy. Slight nystagmus on looking to extreme left. Roentgen ray showed absorption in the frontal region. No signs of hydrocephalus. Basal metabolism, 14.

Diagnosis: Supracellular tumor.

Operation: Transfrontal exploration by Dr. Harvey Cushing. Characteristic pearly tumor about the size of a golf ball in the third ventricle. Removal entirely except for a few small fragments of wall which clung to blood vessels and nerves.

Postoperative Progress: Patient became restless, rolling and tossing constantly, crying and whining incessantly; answered questions only after persistent effort but with perfect comprehension; kept head covered and complained of being cold although temperature remained above normal for nineteen days. The constant involuntary movements of the limbs made it impossible for him to walk. Insomnia was a prominent symptom. This condition disappeared in three weeks. He was discharged well with visual acuity slightly less than before operation.

Histologic examination proved both these cases to be true pearly tumors. (This article will appear in full in *Surgery, Gynecology and Obstetrics.*)

DISCUSSION

Dr. G. B. Hassin said that his examination of Dr. Bailey's slides showed these tumors to be avascular. They probably originated from the mesothelial cells of the pia arachnoid. These cells were also found in the dural interspaces where, according to some authors, they may give rise to tumor formations like endotheliomas. The tumors are benign. It is a peculiar fact that they were never found in the spinal cord or brain tissues proper.

The youth of one of Bailey's patients, 13 years, was noteworthy. The majority of authors assert that these tumors do not occur under 40 years.

He asked whether Chiari's case reported as cholesteatoma of the spinal cord was a real "tumeur perlée" or something else, and whether he had found in the literature a patient as young as his.

Dr. Bailey replied that these tumors usually occurred at the end of the third or beginning of the fourth decade and he thought no case had been reported in a patient as young as 13.

Chiari's report was unaccompanied by histologic examination, hence it was impossible to say whether the growth was a true pearly tumor or not.

THE HISTOPATHOLOGY OF BRAIN ABSCESS, WITH LANTERN SLIDE DEMONSTRATION. Dr. G. B. Hassin.

Published in full in this issue.

DISCUSSION

Dr. Julius Grinker took issue with the speaker on the conclusion he drew from the histopathology of brain abscess as to the value of the Swift-Ellis treatment for neurosyphilis. If Dr. Hall's statement that arsenic had been found in the spinal fluid in 25 per cent. of the cases following intravenous injection were true, the value of the intravenous method was thereby proved—a method which some had considered valueless. However, these findings should not prevent the use of the Swift-Ellis treatment in cases that do not respond to intravenous injections. Some of his results, as well as those of others, had been exceedingly good.

Dr. I. Leon Meyers thought that Dr. Hassin's work showed his statements regarding fatty substances to be true, but this was not absolutely true as regarded every other substance. In Flexner's experiments on poliomyelitis, it had been proved that by injecting the virus into the subarachnoid space of the ape, one could produce the infection, while the cerebrospinal fluid from a patient with this disease proved to be noninfective. It was evident that the virus passes into the brain from the subarachnoid space, but not in the reverse direction.

Dr. G. B. Hassin said he did not intend to discuss the Swift-Ellis method as he believed it had been abandoned by the majority of practitioners. Dr. Hassin's further remarks are covered in the paper.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

The Fortieth Anniversary Meeting, Jan. 15, 1920

George A. Waterman, M.D., *President, in the Chair*

FORTY YEARS' PROGRESS

Dr. Walter Channing

Jan. 6, 1880, when I called the first meeting of the Society to order I little thought that forty years later I would again address it as the only surviving member of the nine then present. That I alone of the nine men should be living supposedly might make me realize that forty years is a great stretch of time.

Fortunately, if the men themselves have gone, we still have the inspiration and comfort of what they stood for and accomplished and younger men to carry the work forward. In 1880 state hospitals in most cases provided good accommodations for their inmates, but in the full sense of the word were not hospitals. They were managed by men who were members of the "Association of Medical Superintendents of American Institutions for the Insane." When an effort was made to change the name in the late eighties to the more modern one it bears at present, the offer was refused in indignation. Not scientific development, but the housing and providing for inmates good care at the lowest per capita cost was the grand object to be achieved. Proper medical attention was regarded as necessary, but careful clinical study of the patient was rare. Laboratories existed for routine pathologic work, There were no training schools for nurses. Mechanical restraint was general and regulated only by the individual superintendent.

In 1879 Dr. Folsom was lecturing on mental diseases in the Harvard Medical School, and in 1883 published a small book for students entitled "Mental Diseases." This book gives a good idea of what might be considered rather progressive ideas for the time. Incidentally it is stated that "in the United States census when there is doubt in the matter the individual is classed as an idiot if the disease occurred before the age of 12 in girls and 14 in boys, and as insane (demented) if above that age."

In 1879 Dr. Jelly left the McLean Asylum and was succeeded by Dr. Cowles. Up to that time the McLean Asylum labored to be a comfortable house for the mentally diseased. The patients were called "boarders" in an effort to make them forget they were under restraint, but the advent of Dr. Cowles meant a step forward. A process of scientific and medical housecleaning began at the McLean which never ceased until he laid down his badge of office, and it was a sad day for Massachusetts and progress in mental diseases when he did so.

I am simply calling attention to a few of the factors which have constituted some of the links in the chain of progress and some of the strongest of these links were forged by Dr. Cowles. He designed the present McLean buildings which have been an object lesson for the whole country. He established laboratories not simply for the study of pathology, which up to his time had been held in leash, but by the deepest and broadest methods the effort was made to solve problems which hitherto had been unfathomable.

The hospital which had now been changed from an asylum underwent changes in administration. The first training school for mental nurses was established and the patients treated as mental cases.

Dr. Folsom was appointed on the Commission by Governor Wolcott in 1896 to consider the then existing methods of supervising the insane. This commission on which Dr. Folsom worked faithfully and energetically may be regarded as the starting point for the care of our insane. This society worked indefatigably, and in 1900 a state board of insanity was appointed.

Our Society was organized at a most fortunate time for the period of stagnation in the study of mental diseases was nearly at an end. Curiously enough, the Guiteau trial in 1882 was a factor in turning the tide. The experts for the prosecution presented a dead wall of conservatism with hardly a chink in it to admit a ray of the light of modern ideas. On the other hand, some of the experts for the defense were reviled and laughed at for some advanced ideas. After Guiteau's execution these ideas rose up and walked abroad uncomfortably like ghosts. It was proved that they were correct and clearly seen that Guiteau was insane, and the subject of a definite form of mental disease. Of course, the Guiteau trial was merely a sign of a general awakening.

Dr. Cowles and other men with advanced ideas needed a forum wherein to present them for discussion and correction. Our Society was intended partly for this purpose. Another most important object was to back up any reform in the care of the mentally ill and we had wonderful opportunities which were largely furnished by the State Board of Insanity through Dr. Copp. He was another of our leaders imbued with what we might call genius. He was constructive in ideas and execution and carried to a successful conclusion reforms and improvements considered impossible.

The greatest thing Dr. Copp did while Secretary of the Board was to work out a plan for the better care and management of our insane. The state care act had been passed and with Dr. Copp acting in cooperation with the Board of Insanity, the backing of this Society and the endorsement of the Massachusetts Medical Society it was possible to impress on the legislature the necessity of going further in the development of our institutions and care of the insane. The Boston Lunatic Hospital was taken over by the State and finally, as the biggest thing of all, the Psychopathic Hospital was built.

What that hospital has accomplished under the influence of the master mind of its director is known, in some measure at least, to us all. The results have been epochal. They have changed an almost unknown branch of medicine into a dignified, scientific medical specialty. They have systematized a mass of disorganized knowledge and in so doing have added greatly to that knowledge. They have improved on treatment and have demonstrated that mental diseases can be cared for as other diseases are in general hospitals, but the greatest result of all has been in the successful efforts of the psychopathic hospital to reach back to the community to attack causes and prevent them. This has been accomplished through the out-patient department. It is evident from the statistics that a large number of persons have been kept out of the state hospitals and many cured before disease has developed. Furthermore, a great fund of knowledge about mental and nervous conditions has been disseminated among patients and their friends.

In time it will come to be seen that an outpatient department like that at the Psychopathic Hospital is of even greater medical and economic value than the hospital itself and an enlightened state will insist on establishing such departments as a means of saving many by diminishing mental diseases.

We cannot use the word "progress" in reference to the period of our Society's existence without thinking of feeblemindedness. Forty years ago it was a neglected and disagreeable subject. Dr. John P. Gray at a meeting of the Association of Medical Superintendents of American Institutions for the Insane in 1884 said: "When we turn to a state like Massachusetts in her philanthropic purposes and see the Governor (Butler) in his annual message declare that the idiots should not be educated because they then come to the realization of their duties and responsibilities which they cannot discharge and that such realization must make them unhappy and that chronic insanity needs no medical care, we might indeed ask if the world was progressing or retrograding."

Think of the change. It is our good fortune to number among our members the man who has been most instrumental in bringing about this great change. The world will rise up and call Dr. Fernald blessed.

Mental hygiene, an entirely new branch of humanitarian work, was unheard of in its present sense forty years ago; it is a wonderful correlation of both social and medical forces, taking advantage of anything and everything, being luckily unhampered by traditions. The National Mental Hygiene Committee in the war will long be remembered. While our Society has not played a leading part perhaps in the Massachusetts Mental Hygiene Society it has contributel some members who have done work which has been really responsible for its success. I would refer in particular to Dr. Stedman.

In time psychiatry will become the most important branch of medical science, including as it does in its scope the moral and physical welfare of the whole human race.

Great progress I believe has been made in our conception of the classification of mental disease. The "key" plan of Dr. Southard can be of great use to the student as he says the plan gives hints for a method of aiming at an eventual diagnosis. It does not prescribe the names of diseases. I believe that we are learning, partly as a result of the war, that labels cannot be pinned on to things as fluctuating as mental conditions. The word "entity," used in regard to mental diseases is a bad one as it implies the idea of fixation. Therapy should always by suggestion indicate that motion must be continuous. Classifications are not of much use if they do not help to keep the patient's mind going along lines which may lead to health, and they are mistaken and therefore pernicious if they convey an idea that a man, by reason of disease, has "lost his mind" and for the time being at least has lost his personality, and become a different kind of being. In the deepest and truest sense mental disease is a result of disturbed social relations of some kind. This is the thing to emphasize when we study treatment and prevention, and it is clearly my opinion outpatient and mental hygiene work which carries us back of the realm of disease and medicine means the next great step in psychiatry.

It is with the greatest satisfaction that one sees on all sides the tendency to drop the word "insanity." We know that it is not a disease but a condition the result of disease, which may affect a person's social status.

I am proud of what has been accomplished in Massachusetts during these recent years, but have fears for the future. In public work requiring the expenditure of money, narrow-minded, short-sighted politicians or managers of institutions often are more influenced by the commercial than the humanitarian aspects of the situation. They must be fought to the last trench unless our progression is to be turned into regression. Here our Society can get in its work. If the hospital policy of the state becomes reactionary, psychiatry is sure to suffer. Eternal vigilance only can prevent it. At our twenty-fifth anniversary I said: "If a medical society has something to do as well as to say, the chances are that it will not only be more useful but will stand a better chance of an active existence." This is as true today as in 1905.

IN MEMORY OF FORMER MEMBERS

Dr. Edward W. Taylor

Dr. Taylor spoke briefly of the activities of the members who had died since the thirty-fifth anniversary, which was held in the same club dining room in 1915. He spoke particularly of Dr. Henry C. Baldwin, Dr. James J. Putnam and Dr. August Hoch.

ENDOCRINOLOGIC ASPECTS OF SOME NEUROLOGIC CONDITIONS. Presented by Dr. Walter Timme.

THE MODERN SPECIALIST IN UNREST

Dr. Southard spoke of the social conditions with which we are confronted in these postbellum days. He emphasized the fact that many of the labor troubles are attributable to leaders of a certain mental type rather than to insurmountable social complications. He spoke of the development in social fields of psychologic and mental examinations and what tremendous aid can be gained in industrial problems when the right men are fitted into the right situations. He felt that the development of psychiatry and psychiatric social service would open unlimited fields of service in the community and the nation. Just as specialists in accountancy and economic efficiency have developed, he felt that a field for the psychiatrist in promoting efficiency along lines involving psychiatric problems was developing.

PHILADELPHIA NEUROLOGICAL SOCIETY

Jan. 23, 1920

J. Hendrie Lloyd, M.D., *President, in the Chair*

STUDIES ON A CASE OF HYPOPITUITARISM. Presented by Drs. E. A. Strecker and H. S. Newcomer.

Family History.—The father of the patient was intemperate; the mother alcoholic, immoral and possibly feebleminded. One sister, aged 4, was "backward," while three brothers and one sister appeared to be normal. Neither of the parents nor any of the siblings showed unusual physical characteristics and were of average stature and weight. An almost indescribable condition of squalor, filth and utter neglect was found in the home. The patient was then 10 years old and had never attended school.

Personal History.—When the patient was 10 years and 3 months old, the height was 61 inches, the weight 109 pounds; ten months later she weighed 165 pounds, had gained 2 inches in stature; the waist measured 33 inches and the bust 38 inches.

June, 1918: "The girl's appetite is enormous. She consumes half a loaf of bread at one meal and requires several helpings of everything on the table. She is gluttonous in her manner of eating, forgetful, and sluggish in her movements."

April, 1919: "Growing very rapidly, appetite is gluttonous."

October, 1919: "Appetite abnormal and bolts whatever is given her; very sluggish in all her movements; likes to go to bed early and sleeps late and soundly."

During the two months prior to her admission to the hospital there was evidently some improvement. The patient became somewhat brighter and more interested, but was still slow, plodding and indifferent.

Physical Examination.—At the time of the physical examination the patient's actual age was 12 years, 2 months; apparent age about 17 years. The height was 63 inches; weight, 155 pounds; head rounded, circumference 22 inches. The profile showed slight maxillary prominence. The nose was somewhat saddle-shaped and short. The ears were large with fleshy lobules. The teeth showed moderate spacing with protrusion of the upper central incisors. The neck was short—girth 14 inches. The hands were wide and relatively short with a tendency to slight finger tapering. The shoulders were rounded and fairly broad. The chest was broad and deep—circumference 37 inches above and 38.5 inches over the breasts, which were well developed, firm and globular with clearly defined pigmented areola about the nipples. The pelvis was straight with a tendency to the masculine type—circumference at hips, 40 inches. Upper extremities were feminine in type—girth of the arm was 11 inches, forearm 10 inches. Lower extremities were of the feminine type—girth of the thigh was 25 inches, calf 14.5 inches.

The anthropologic measurements on the skeleton were: total heights, 156 cm., height of the acromium process above the floor 130 cm., the proximal end of the radius 97.5 cm., the styloid of the radius 77 cm., the tip of the finger 59.5 cm., the umbilicus 97.5 cm., the anterior superior spines 94 cm., the upper border of the symphysis 85.5 cm., the proximal end of the tibia 46.3 cm., the internal maleoli 6 cm. The interacromial distance 33 cm., inter-anterior superior spinous 21 cm., and the span 164 cm.

Hair: The head hair was medium fine, dark brown, rather scant and growing low over the forehead and in the temporal region. The eyebrows were scant. There was no other hair growth except a small amount on the labia majora and a scarcely perceptible down on the lower limbs. December, 1919, the menses appeared for the first time.

The skin was dry, warm and slightly roughened and thickened.

Adipose Tissue: There was a large amount of firm fat, more or less evenly distributed with some excess in axillae, breasts, over abdomen and in the gluteal region. The fat had no myxedematous characteristics.

The temperature was continuously subnormal, often as low as 96 degrees. The pulse, respiration and all other examinations yielded normal results. The roentgen-ray report showed an approximately normal sella. The radial epiphyses were open.

Eye examination showed normal fundi; some constriction of the form field especially for the left eye and the slight overlapping of the color fields.

There were no psychotic symptoms. The mental age was 7.5 years, being a retardation of 4.5 years.

The patient while not active or alert was neither dull nor sluggish. She showed a fair degree of interest in various occupations and did well under supervision. She now ate only an average amount. As much as 400 gm. of sucrose was given without producing glycosuria and with a 40 mg. fall in the blood sugar; 325 gm. of glucose did not result in a glycosuria and produced a fall of 15 mg. in the blood sugar. The following experiments were made to show the relationship of the sugar tolerance to the secretions of the endocrine organs. The ingestion of 225 gm. of glucose with the simultaneous subcutaneous injection of 2 c.c. of pituitary extract produced within an hour a blood sugar rise from 128 mg. to 256 mg. per 100 c.c., and an output of sugar in the urine of 0.9 gm. Within two hours the blood sugar fell to 148 mg. The blood pressure remained unchanged. With the usual breakfast (toast and coffee) and two hours previous to the ingestion of 225 gm. of glucose, 20 grains of thyroid extract were administered by mouth. The blood sugar at the end of one hour was 149 mg. per 100 c.c. The average for the patient as frequently determined was not more than 135. There was no glycosuria. The injection of 2 c.c. of 10 per cent. boiled anterior pituitary lobe extract produced no thermic response.

The Goetsch test gave a blood pressure rise of 15 mm. without glycosuria. During a two hour period there was an increase in white blood cells from 13,000 to 14,900 affecting the neutrophils. A 5 mg. dose of epinephrin resulted in a leukocytic increase from 11,575 to 27,000 also neutrophilic, the blood pressure rose from 130 systolic to 175 systolic during the first hour and there were slight subjective symptoms of cardiac discomfort. The urine contained 3,375 gm. of sugar. The subcutaneous injection of 1 mg. of neutral sulphate of atropin produced moderate pupillary dilatation and accelerated the pulse from 100 to 115 over a period of several hours.

The administration of 0.01 gm. of nitrate of pilocarpin resulted in a scarcely perceptible supra-orbital sweating and possibly slight salivation.

The basal metabolism as determined by Dr. Jonas gave the following figures: respiratory quotient 0.85, total calories per hour 90.74, total calories per square meter per hour 53.7. The latter figure is 7 above the normal for a girl of 12, 14.5 above the normal for a person of her maturity.

Discussion.—The patient showed neither vagatonic nor sympathicotonic symptoms nor signs. She had, however, a syndrome of skeletal overgrowth, pathologic adiposity and unusually high sugar tolerance; the latter could be promptly decreased by the administration of pituitary extract but not so definitely by thyroid extract. Neither the character of the adiposity nor the appearance of the skin were suggestive of hypothyroidism and no other signs with the exception of the high glucose tolerance indicated the possibility of such a condition. The adiposity was more or less generalized. It had resulted in a recent increase in weight of 55 pounds and a present weight of 155 pounds, which was 82 pounds overweight for her age and 53 pounds overweight for her height. Referring to the Prudential Life Insurance Company tables, girls of her height and of an age, 3 to 7 years greater, weigh 35 pounds less. The highest normal weight for women of this height at any age is 14 pounds less. There is, however, a definite skeletal overgrowth. Aside from a 2 inch increase in height during a period of ten months, we have to consider certain skeletal measurements which compare in an interesting way with the normal for our patient's age. The ratio of the height of the symphysis to the total height is 54.8 per cent., being 4.8 per cent. greater than normal. The ratio for the umbilicus is 62.5 per cent., a figure which is not reached in any race. The length of the arm is 70.5 cm., 9 cm. above the normal and the ratio to the height is 42.2 per cent., 1.7 per cent. above the normal. The span is 2.25 cm. above the normal. The ratio of the span to the height is 105 per cent., 5 per cent. above the normal. There was, therefore quite definitely, in addition to a generalized overgrowth, a relative increase in the length of the long bones. If the pituitary disease had occurred later in life or after epiphyseal union, the result would have been the development of an acromegaly instead of gigantism.

Certain minor physical characteristics of some interest were the spacing of the teeth, the low temporal hair growth and the slightly tapering hands.

In connection with the negative roentgen-ray findings and other neighborhood signs, Cushing (*Am. J. Med. Sc.* **64**:313, 1913), and more recently Timme (*J. Nerv. & Ment. Dis.* **1**:460, 1919), have both called attention to the not infrequent occurrence of a normal sella in pituitary disease of a type similar to the one here presented.

The child had developed something which simulates gigantism and is succeeded by adiposity together with unusual sugar tolerance without neighborhood signs. It may be assumed that there was a hyperfunction of the anterior lobe, which, prior to epiphyseal ossification, resulted in skeletal overgrowth. The present posterior lobe deficiency completes the syndrome and accounts for the high sugar tolerance, the adiposity, the subnormal temperature, the somnolence and the dry skin.

These cases are important because they present a definite indication for treatment. The administration of the extract of the whole gland has been commenced with the intention of increasing the dose until the sugar tolerance is normal.

DISCUSSION

Dr. J. Hendrie Lloyd referred to an unfortunate experience with a case of dystrophia adiposo-genitalis in a man 22 years of age, who had attained a weight of 230 pounds, who had arrested sexual development, beginning optic neuritis and a larger sella turcica. Dr. Lloyd had him under observation for a few weeks. He was not quite so thoroughly studied as the patient of Drs. Strecker and Newcomer, but the glucose reaction did not show increased tolerance. He began to have curious epileptiform attacks and developed severe headache. The case seemed to be such an unmistakable one that Dr. Lloyd had him transferred to Dr. Frazier, who agreed with the diagnosis and decided to operate. The first incision was made—the bone had not been lifted—when the patient had symptoms of respiratory failure. He was kept alive for ten hours, with artificial respiration. Unfortunately, necropsy was not obtained. It was extraordinary that ether narcosis produced respiratory failure so quickly. As necropsy examination could not be made the diagnosis was not fully confirmed.

Dr. Francis X. Dercum said that in these remarkable cases of endocrine disorder it appeared that more than one gland is affected. Dr. Strecker had beautifully demonstrated his case to be one in which the pituitary gland was affected. Dr. Dercum questioned whether it was the only one; whether other glands did not react in some way. Sometimes a hint is given by the menstrual function.

A FORM OF BRACHIAL PLEXUS PARALYSIS CAUSED BY A BONY GROWTH OF THE UPPER THORAX RESEMBLING THAT CAUSED BY CERVICAL RIBS. Presented by Dr. William B. Cadwalader.

Dr. Cadwalader said that this condition did not appear to be generally known; similar cases do not seem to have been recorded.

In addition to the signs of irritation and compression of the trunks of the brachial plexus, there were also signs of compression of the blood vessels. The course of the disease has been slow, but gradually progressive. In Case 1 only slight progression had occurred in fifteen years, and in Case 2 there was no progression until after five years. The nature of the bony growth has not been determined, but its effects were striking and resembled those produced by cervical ribs. Unlike that condition, however, the pressure was exerted from below. The disease was diffuse, attacking more than one rib at a time, and the costal articulations as well. In Case 1 the disease was bilateral; in Case 2, unilateral. Syphilis was not present. It was significant that in Case 1 there was a clear history of four distinct attacks of pneumonia, and permanent evidences of an infectious process still remained in the lungs. Dr. Cadwalader believed that a chronic infectious process was the most probable cause of the bone disease in both cases.

Case 1.—*History.*—P. F., aged 55, admitted Nov. 25, 1919, complained of precordial pain which extended down the left arm.

In 1903 he first noticed weakness in his right hand. There was no pain. The weakness slowly progressed and extended, involving the muscles of the forearm and arm and then remained stationary. There was still considerable voluntary power. Since an attack of influenza a year ago, he had had precordial pain which radiated down the left arm into the hand and into the upper part of his chest. It was not increased by exertion but was occasionally sufficiently severe to keep him awake at night. He had had four attacks of pneumonia. For many years he had been exposed to cold and wet, and had used much alcohol.

Physical Examination.—Muscular development was rather poor. The right upper extremity was moderately wasted, particularly the muscles of the right hand and forearm. The muscles about the shoulder girdle were similarly affected, and to a lesser degree the corresponding muscles on the left side. The muscles of the left hand and forearm were normal. There was distinct weakness of the right upper limb, most marked in the distal portion but there was still considerable power. No paralysis was present on the left side. There were some fibrillary or fascicular contractions in the triceps of the right side. Sensation was normal. The biceps and triceps jerks on the right side were distinct, but less than on the left. The fingers of the right hand were held in slight extension at the first phalanx and partial flexion of the second and third phalanges. There was limitation of motion of the fingers. Flexion and extension were weak.

The lower extremities were entirely normal. The blood count was normal and blood Wassermann reaction was negative. Expansion of the chest was limited on the right side with increase of vocal fremitus and decreased resonance, especially over the upper lobe. At the right apex there was bronchial breathing and a few râles and at the right base a friction sound. The diaphragm on the right side was fixed; on the left it moved freely. There had probably been a chronic tuberculous process in the right upper lobe with retraction and probable cavity formation. The right basal pleura was thickened and the diaphragm was fixed there with compensatory emphysema on the left side. The heart was normal. There were varicose veins over the right upper thorax and shoulder, and on palpation the first rib and the right shoulder were rough and considerably thickened.

The roentgen-ray examination showed signs of an old tuberculous process of the upper lobe of the right lung with cavity formation and marked fibrosis pulling the arch and the trachea to the right with fixation of the outer aspect of the right diaphragm. The first rib on the right side and the third rib on the left side were greatly thickened. All other examinations were negative, except for marked infection of the mouth and teeth.

Case 2.—*History.*—C. R. E. In 1913, after using a wrench with his right hand, the entire upper limb became swollen. At the same time the hand and forearm felt numb and the fingers became strongly flexed on the palm of the hand, and the hand flexed on the forearm. He could readily straighten the fingers with the left hand, and they remained extended. After use, the right upper limb grew progressively weaker, and the swelling and discomfort continued, but after rest these symptoms entirely disappeared. The difficulty was greater when the limb was held extended over his head, as in hanging pictures. He had never had pain until a week before.

Physical Examination.—The right upper limb alone was affected. Grasp of the right hand was at first as good as the left, but became weak after continued effort. The biceps and triceps jerks were slightly exaggerated on the right and normal on the left. Sensation everywhere was normal; there was no muscular wasting. There were marked varicose veins over the chest, particularly on the right side, and also in the upper portion of the right arm and shoulder. Recently he had had attacks in which he had difficulty in turning his head from left to right, associated with a feeling of faintness. Otherwise physical examination was negative.

A roentgen-ray examination showed premature ossification of the costochondral articulation of the second rib of the right side. At this point there was a fairly large osseous growth and ossification of the costochondral articulation of the first rib with some bony proliferation. External to this articulation the first and second ribs were fused together. The sternal end of the clavicle was slightly larger than its fellow, but the difference was not marked.

ON SOMATIC SYMPTOMS IN NERVOUS AND MENTAL DISEASES, WITH THE REPORT OF AN INTERESTING CASE. Presented by Dr. Francis X. Dercum.

Somatic or visceral phenomena in nervous and mental diseases present themselves under three different conditions. First, they may be directly due to and symptomatic of the underlying nervous affection. For example, in neurasthenia the deficient innervation and lessened inhibition present may give rise to characteristic phenomena presented by the circulatory apparatus, the digestive tract, the sexual apparatus or by various glands. In hysteria, the list of visceral phenomena that may be present is large: anorexia, vomiting, tachycardia, vasomotor phenomena, cough, rapid breathing, aphonia, polyuria, anuria, phantom tumor and especially local pains which lead to abdominal and other unnecessary operations. In a case presented some years ago, because of insistent complaint of pain, the left testicle, apparently entirely normal, had been removed. The pain, hypochondriac or, to speak more accurately, hallucinatory in character, at once transferred itself to the remaining testicle. Similarly much attention is at times given, especially in unrecognized cases of hypochondria, to the eyes, the nose, the throat, the digestive tract and other organs. I may mention a woman who had had thirteen pairs of eye glasses, twenty odd sets of false teeth, and who finally had had her stomach scrubbed out by the method of Dr. Turck, all to no purpose.

The second condition is one in which nervous symptoms are present but are secondary to visceral disease. For instance, general weakness sooner or later supervenes on visceral disease but this weakness differs radically in its symptomatology from that of neurasthenia, and is especially featured by the pains and other phenomena which direct attention to the structures involved or it may be to the general morbid process at work. Again, the exhaustion and the toxemia the result of somatic disease may in given instances give rise to mental disturbances, confusion, stupor or delirium.

A third condition encountered—but on the whole infrequently—is one in which primary nervous or mental disease and primary visceral disease coexist in the same patient; for example, brain tumor and hysteria, pelvic disease and hysteria. Here the situation may at times be misleading and difficult to unravel.

History.—H. S. T., aged 21, single, white, sailor, was admitted to the Jefferson Hospital, July 10, 1917, complaining of difficulty in swallowing. The family history was negative.

Personal History: The patient denied venereal infection and used neither alcohol nor tobacco. He had had an intermittent discharge from the right ear in childhood, a convulsion at 6 years of age, and two more at 9 years. According to his unsatisfactory account, these began in the right index finger, and he was unconscious afterward for several hours.

In 1914, while at work, his sweater caught on the shaft of a donkey engine and he stated that he was turned around the shaft five times and sustained a 4 inch scalp wound over the left temporal region. His right ankle also was hurt.

Two years before admission he had two convulsions, one day apart. Each lasted about an hour and a half and was followed by many hours of unconsciousness; sometimes he said two, sometimes four. He said that he bit his tongue.

Present Illness.—One year ago while eating, he felt a contraction in his throat which he thought was the beginning of a convulsion. He went into the open air, but returned in a few minutes and finished his meal, though he did so with much effort. He had difficulty in swallowing and this difficulty in swallowing, he asserted, had persisted ever since. He stated that it was especially difficult to swallow solids and that swallowing was accompanied by much belching and often by regurgitation of food. He had lived for a long time mainly on milk and eggs. He was constipated and suffered from headaches.

Condition on Admission.—Esophagoscopy by Dr. Chevalier Jackson, disclosed a superficial erosion in the upper third of the esophagus contraindicating insertion of the tube beyond this point. Dr. Jackson suggested that a second esophagoscopy be done later.

Because of the occurrence of two convulsive attacks while chewing food at breakfast, the patient was afraid to masticate thinking it would bring on a convulsion. He had therefore been bolting his food and this was attended by a special effort which brought on some sensation of distress or possibly a spasm of the esophagus. The act was further followed by much regurgitation and belching.

Roentgen-ray examination was negative. Wassermann and other clinical examinations were negative.

Treatment and Course.—Under suggestion the patient was gradually persuaded to chew his food properly and he swallowed without apparent difficulty No convulsions were induced. Regurgitation and belching were also absent. The case was regarded as probably hysterical in origin, although other hysterical stigmas could not be elicited. The patient was later placed on ordinary house diet, ate normally and was apparently relieved. On July 23, 1917, he was discharged.

During his stay in the ward he was noted as rather peculiar. He was fault-finding and disagreeable; at times sullen and insubordinate; at times captious and complaining about his food. While these facts were noted at the time, no special emphasis was laid on them. No convulsions were observed at any time during his stay at the hospital.

He was readmitted on Oct. 27, 1917, again complaining of difficulty in swallowing. He stated that while chewing his food his throat felt as though it were swelling. He had again had special difficulty in swallowing solid food. The sensation of swelling in the throat passed away after taking a sip of water or after actually swallowing the food. He also complained of regurgitation.

Physical examination resulted negatively. Esophagoscopy by Dr. Jackson on Nov. 15, 1917, failed to reveal any erosion, stricture or spasm of the esophagus. As before, the patient was treated by suggestion and as before successfully. As before it was noted that he was a little peculiar. While he finally admitted that he no longer had difficulty in swallowing, he insisted that he had regurgitation. This, observation failed to confirm. It was now especially noted that he was reserved, uncommunicative, and suspicious in manner and introspective. He would ask the meaning of medical terms which he had heard used in connection with his case and was quite disturbed by the word "functional." It was noted also that he frequently busied himself writing on small pieces of paper, fragments of envelopes and other odds and ends. On one occasion with much solemnity he handed to one of my assistants an envelope, containing minute clippings of newspapers, a blade of a safety razor, the stump of a lead pencil and pieces of paper containing much closely written matter. The latter when examined consisted of a rambling and disconnected account of his symptoms and feelings in which a distinctly paranoid attitude of mind was discernible. He now also began talking of various strange sensations which he referred to the upper portion of his abdomen. His descriptions and the fact that the examinations of the abdomen resulted negatively indicated visceral hallucinations.

He left the hospital on December 7. His conduct continued to be peculiar. He was uncommunicative and solitary. He refused to eat the food provided by his aunt, and to which according to my inquiry there could have been no reasonable objection, but purchased his meals outside at various cheap restaurants. He offered no explanation, and it is not improbable that his conduct was the outcome of delusions, possibly having their origin in various visceral hallucinations. The latter he continued to refer to the throat and to the abdomen. Hallucinations of taste also appear to have been present.

Diagnosis.—The convulsive seizures which were neither reconcilable with those of hysteria nor of true epilepsy, remained unexplained. It was only the later appearing mental features which led to a correct appreciation of the case. Evidently the case was one of dementia praecox in an early stage, the symptoms of which at the time he first presented himself were just beginning to reveal themselves. The convulsions are to be regarded as among the epileptiform attacks — the motor crises — at times encountered in cases of dementia praecox, especially in the developmental period. The case teaches a valuable lesson as to the interpretation of local or visceral symptoms, when the latter have no or little physical foundation, and when, still further, hysteria offers an inadequate and unsatisfactory explanation.

PATHOLOGIC LYING IN A CRIMINAL. Presented by Dr. N. S. Yawger.

Dr. Yawger, after brief review of the meager literature, reported a case in which there was shown the marked character deviation of a pronounced and persistent fabrication, abnormal in type; this was a degenerate condition and was almost invariably accompanied by other forms of degeneracy.

In addition to the pathologic type of falsification the man was guilty of two attempts at homicide, one successful; three attempts at suicide, probably rape and possibly simulation of insanity. Subsequently, the convict was placed in the Pennsylvania Eastern State Penitentiary, where he is undergoing life-sentence.

The man, a young Lithuanian, while in the service of the Russian guard, became infatuated with a girl, whose father objected to the attachment because of a difference in religion. The man then attempted to kill both the girl and himself; they recovered. She fled to America but he followed, where, after ravishing her as she charged, he later murdered her.

Dr. Yawger remarked that the prisoner was without positive anatomic stigmas. In appearance, he was tall, erect and finely proportioned; his head was well shaped and his countenance was rather inviting; he was polite, quick-witted, of the utmost assurance, an incessant talker and with a great deal of general but superficial knowledge which he used with considerable linguistic ability.

As to his fabrications, his most remarkable invention was while awaiting trial in Moyamensing prison. It related to another atrocious murder in which the prisoner said he was implicated and which he attempted to tell a priest but later refused to confess him until the authorities had been apprised of the crime. He then reported that he had stood by while one man murdered another, that he saw the grave dug and the body placed therein. A detective was detailed to investigate the case but found that the presence of water rendered it a physical impossibility to have dug a grave there and it was concluded that the prisoner was simply "exercising his imagination." He still recalled the circumstances and regarded it as a good joke.

The prisoner was suspicious and was constantly making accusations against overseers and cellmates. He frequently reported that cellmates were secreting in their cells coffee, cocoa, sugar and money, all of which were contraband. He once charged that another prisoner was going to stab him with a pair of shears and at another time that two convicts were trying to cut through their cell door. With grave concern, he once reported that an inmate was hiding tools in a sewer, preparatory to an attempt at escape; on investigation, it was found that the cover to the sewer was securely cemented and had been so for years.

When pressed for an explanation he stated that "I was bluffing;" that "others have lied to me first;" that "often I lied to defend or to protect myself which I considered a justifiable fraud;" and that "sometimes I have lied to develop a situation about which I was suspicious."

The mental classification of the man is psychopathic personality. The outstanding features are: emotional instability, abnormal sexuality, pathologic lying, possibly still suicidal.

THE CURATIVE INFLUENCE OF INFLUENZA ON A CASE OF SPECIFIC MENINGO-MYELITIS WITH CYSTITIS. Presented by Dr. C. W. Burr.

This paper was published in the May number of the Archives of Neurology and Psychiatry, page 536.

DISCUSSION

Dr. Alfred Gordon said that about a year and a half ago he was called to see a case of paraplegia of long standing with much pain and bladder incontinence. The history suggested syphilis and the patient was given ten injections of neo-arsphenamin. The bladder disturbance improved and she began to move her limbs slightly. Suddenly she developed bronchopneumonia of great violence; the temperature reached 104 degrees. When she recovered from the bronchopneumonia, Dr. Gordon noticed immense improvement in the condition of the limbs, and at present the patient can walk a little.

Dr. William B. Calwalader said that cystitis retarding the return of power in paraplegic cases had been noticeable in cases of paralysis from gunshot wounds of the spinal cord observed in France. The work of Head and Riddock and others had demonstrated that return of function was almost nil unless cystitis could be cured. The importance, therefore, of the utmost care in the management of the paralyzed bladder cannot be exaggerated. Dr. Cadwalader also said that return of function after the administration of antisyphilitic treatment can be long delayed; function may reappear long after treatment has been stopped. Dr. Spiller had reported such cases.

Dr. Dercum thought that all were familiar with the results of infection in paresis. Pilcz of Vienna had brought about striking remissions by inoculating the patient with the virus of erysipelas. He thought Dr. Burr's case was unique.

Dr. C. W. Burr said there was one point he might not have emphasized sufficiently; namely, for six months after the woman left the hospital she received no antisyphilitis treatment of any kind. He was quite convinced that in paraplegics from all causes the cystitis was certainly a measure of loss of power. This was the only case of this kind he had ever seen, but he was convinced that if a patient had severe cystitis and that cystitis improved tremendously there would be some improvement in the legs. Dr. Burr said he had seen this result a number of times.

MONOPLEGIA SPINALIS SPASTICA. Presented by Dr. William G. Spiller.

This paper appears in full in this issue.

DISCUSSION

Dr. Charles S. Potts reported the following case: A man, 48 years of age, for the last two years had had purulent bronchitis, no tuberculosis. About six weeks ago he developed pain and weakness in the left leg. He now had some atrophy of the muscles of the thigh and distinct pain and tenderness in the left knee. The pain extended up the leg toward the hip. He had some fibrillary twitchings in the thigh muscles. Electrical reactions were normal. There was marked increase of the knee jerks, more so on the left, and ankle clonus on both sides. At times he had some fever. The sphincters were normal and roentgen-ray findings were negative. Dr. Potts said the man probably had a toxemia of some sort, possibly due to the purulent bronchitis and that he might have arthritis in spite of the absence of swelling and roentgen-ray findings. Possibly it may be a case like Dr. Spiller's, in which the roentgen ray failed at first to reveal anything.

Dr. Woods said that probably most of those present knew of the existence of these commissural fibers, which, according to Obersteiner, pass over as a regular constituent of every motor root, spinal and cerebral, and thus bring the muscles corresponding to each root under the influence of the motor cells of the same level in the opposite side; but that the Society was indebted to Dr. Spiller for showing this example of the probable functioning of such fibers. If a particular muscle receives its chief innervation from a certain segment, it is believed that after the destruction of the cells of that anterior horn other cells in neighboring segments above and below on the same side may assume control of the muscle. Dr. Woods understood from Dr. Spiller that in this patient the lesion had extended so far in a vertical direction as to remove this possibility and therefore he could see no other explanation of the existing spasticity except the one Dr. Spiller had proposed.

PRESIDENT'S ANNUAL ADDRESS. Delivered by DR. J. HENDRIE LLOYD. This paper will be published in full in an early issue.

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Seventy-Ninth Regular Meeting, March 2, 1920

WALTER TIMME, M.D., *President*

PRESENTATION OF CLINICAL MATERIAL. CHRONIC NONDEGENERATIVE HEREDITARY CHOREA. Presented by DR. I. S. WECHSLER.

This patient showed a clinical picture closely resembling Huntington's disease, but certain distinctive features suggested that it might be a distinct clinical entity. A married woman, aged 36, had had peculiar movements of arms, hands, body and legs and twitchings of the face for about sixteen years, gradually increasing in intensity for a time, then remaining comparatively unprogressive. A slight weakness of the heart and faintness were complained of at the onset, which is said to have followed a miscarriage. There were no convulsions, biting of the tongue, amnesia, etc. The attacks were closely linked with the patient's emotional state, suggesting a possible hysterical condition. The patient's father, who was the uncle of her mother, also suffered from chorea for twenty years. One brother had shakings. Of her children, one daughter was not nervous, but had poor eyesight and nystagmus; the second daughter had twitching and attacks of weakness. The shaking was not choreic. Two small boys both had chorea and nystagmus.

The patient showed a number of abnormal involuntary, irregular, jerky purposeless movements of whole parts—arms, legs, body, hands, and twisting of the whole body. The eyeballs wandered in irregular fashion. All these movements were intensified by emotion while control inhibited them for only a short time. The movements were in general more rapid than in chorea. No pathologic reflexes were found. Vision and hearing were normal, except for the choreic nonrhythmic movement of the eyes. Mental status was perfectly normal, a slight tendency to forgetting probably being due to lack of attention.

The oldest boy, aged 9½, at 7 years developed a condition diagnosed as acute chorea. The condition improved somewhat after six months. At 7½ years there was a second acute attack which still continues.

The younger boy, aged 6, has had slight twitchings since he was 3 years old. Slight unsteadiness in equilibratory and nonequilibratory tests, of a choreic nature, was found on examination. Some nystagmus on looking forward and trying to fix the gaze was noted.

Unlike Huntington's chorea, the onset of the attack was at the early age of 20. The movements are quicker, the face shows more grimaces, speech is differently affected, somewhat forced and slow but not scanning. The gait is clownish. Mental degeneration is absent. Hysteria might be adduced as a cause, especially hysteria associated with chorea, while other forms of chronic chorea, such as chorea gravidarum and paramyoclonus multiplex, have some features suggestive of this case, but do not correspond sufficiently to warrant the diagnosis. The point of particular interest is that it is a nondegenerative, nonprogressive type of hereditary chorea.

DISCUSSION

Dr. Sylvester R. Leahey of Brooklyn stated that he had seen conditions similar to that of Dr. Wechsler's patient in women of about 50. These had paranoid and suicidal tendencies. In view of the youth of the patient, he thought that the mental symptoms that could not be observed now, might develop later.

Dr. J. H. Leiner did not believe that this case came under the true category of Huntington's chorea, since the choreiform movements were entirely too lightning-like in character.

HYPERTHYROIDISM IN A GIRL OF NINE YEARS OF AGE. Presented by Dr. Morris H. Frantz (by invitation).

This case was of interest because of the infrequency of the condition in children. When the patient came to the Neurological Institute Clinic, she was fidgety, would develop rages and had palpitation on violent exercise. Muscular sthenia, ocular manifestations and a distinct exophthalmus were present. Tachycardia and slight tremor of the hand were also noted. The mental age was 12½ years.

The patient's father had rheumatic arthritis, the mother suffers from hyperthyroidism. Goiter was present in a maternal aunt. The child was born in a town in Germany in which goiter was prevalent. The mother developed goiter at the time of the child's birth. The same condition was diagnosed in the child when 1½ years old. The condition became aggravated at the time of the emigration of the family to America during the submarine blockade.

DISCUSSION

Dr. Walter Timme called attention to other glandular stigmas present, and questioned whether the case could be called one of hyperthyroidism since hypothyroidism and hyperthyroidism and hypoadrenalism all appeared to have been present at some time. The absence of lateral incisors, and the abnormal configuration of the teeth were of particular interest for the glandular study.

Dr. J. H. Leiner asked whether this case might not be considered a fruste type since the patient was well preserved and did not show any of the cardinal symptoms of an advanced thyrotoxicosis.

Dr. Frantz said that hypothyroidism and other pluriglandular syndromes had been apparent in the patient, but that since the hyperthyroidism was the prominent feature he had placed special emphasis on that condition.

ACUTE INFECTIOUS MYOCLONUS MULTIPLEX AND EPIDEMIC MYOCLONUS MULTIPLEX. Presented by Dr. J. Ramsay Hunt.

Dr. Hunt called attention to the problem of localization of acute infections in some part of the nervous system. The varieties of clinical types in Heine-Médin's disease, for instance, emphasized that certain strains of the same infective organism might have special affinities for certain tissues of the nervous system, and thus bring about the special clinical type of reaction.

Such special forms of localization of acute infection are to be found in acute infectious myoclonus multiplex and epidemic myoclonus multiplex. The form is characterized by lancinating pains, muscular contractions and twitchings and a delirium of toxic origin. This group of symptoms constituted a well defined clinical type of neural infection which differed from those previously recognized and was encountered both in sporadic and epidemic form.

The onset of the disease is acute and is characterized by shooting pains of great intensity in the trunk and extremities. Spinal pains are sometimes present. The pains are followed by characteristic muscle jerks, waves and twitchings (myoclonus multiplex, myokymia and fibrillary contractions). The contractions make their appearance first in the parts in which the pains were first felt. A week may elapse in some cases between the appearance of the pains and the myoclonus and myokymia. The twitchings are bilateral, multiple and may become generalized. There is sometimes a tendency to localization in certain regions of the body, especially in the abdominal musculature. The contractions are quick and of short duration, individual muscles or portions of muscles are involved, but not synergic groups. Slight movements of the toes, fingers and extremities may occur in severe myoclonic twitchings, but never to the extent found in chorea or cortical myoclonia.

There is usually a moderate fever. In some cases which proved fatal the temperature rose in the later stage of the disease. An acceleration of the pulse rate was noted, and in most cases, a delirium which varied in duration and intensity with the degree of infection. There is often marked hyperidrosis, and the degree of sweating seems to bear some relation to the activity of the myoclonus phenomena. There is no paralysis or paresis of any muscle or group of muscles; no anesthesia is encountered, with the exception of occasional transient areas of hypalgesia. No ataxia and no loss of deep sensibility; tendon reflexes are present and active. Rarely the knee jerks may be diminished and the Achilles jerks are absent during the height of the disease. The cranial nerves show no evidences of involvement except for the myokymic twitchings. The optic nerves are normal. Skin reflexes are present and equal (no Babinski sign). When abdominal myokymia was present, the abdominal reflexes were exaggerated.

Dr. Hunt had observed twelve cases of this type in sixteen years; two cases were seen more than ten years ago, and the remaining ten within the last three months. The first cases were evidently sporadic, the latter epidemic. The distinguishing features, acute pain of lancinating variety, with muscular waves and twitchings were always present. Delirium was present in eight of the cases. The myoclonus delirium was a characteristic toxic delirium with hallucinations, illusions and transitory delusions. Restlessness, insomnia, apprehension, disconnected thought and mental confusion were present. Apathy and a tendency to stupor were sometimes encountered in the late stage. In the four cases without distinct delirium there was insomnia, restlessness, irritability and excitement in the early stage and later a tendency to apathy and dulness.

That the disease is infectious is evident from the character of the onset, the fever and delirium. Multiple neuritis and acute poliomyelitis may be excluded as diagnoses since the paralysis or weakness of the muscles associated with these diseases is absent. There is no appreciable tenderness along nerve trunks. Dubini's disease may also be excluded since it involves paralysis. Epidemic lethargic encephalitis is an especially interesting possibility.

The epidemic myoclonus multiplex probably belongs to this group and represents a special myoclonus type of this affection. The infectious agent of epidemic encephalitis and epidemic myoclonus multiplex is apparently the same.

The motor and sensory symptoms of the disease are only irritative in character, in spite of the severe and sometimes lethal infection of the nervous system. There is no paralysis or anesthesia, and this fact gives the disease an added interest, since the myoclonus syndrome is not found in other forms of spinal and neural infections.

DISCUSSION

Dr. Charles Rosenbeck asked whether any of the patients with myoclonic phenomena had subsequently developed lethargic encephalitis. In three cases, which he had observed, the violent neuralgia, myoclonus, mild temperature and mental confusion were the dominating symptoms. There were no demonstrable sensory changes except dysesthesia. One of these patients recovered in a short time, without further incident. The others, however, after a variable time, gradually became merged into the syndrome of lethargic encephalitis.

Dr. C. C. Beling of Newark reported that he had seen five or six cases of similar sudden onset of pain, occupational delirium and muscular incoordination.

Dr. William M. Leszynsky had seen several cases of encephalitis in which there had been clonic spasm of the abdominal muscles. Another patient developed a clonic spasm of the diaphragm without hiccup.

Dr. M. Neustaedter reported three cases, seen within the last four weeks, of undoubted lethargic encephalitis complicated with myoclonus involving the abdominal muscles in two cases and the facial muscles on the left side of the face in one.

Dr. Gregory Stragnell (by invitation) asked whether hiccup was a feature of the symptomatology observed by Dr. Hunt. From Canada it was reported that 50 per cent. of the persons suffering from encephalitis had a hiccup that persisted for seven or eight days.

Dr. J. H. Leiner had had a case with diplopia. The diplopia developed ten days after the beginning of the disease. It was diagnosed as lethargic encephalitis. This patient had choreiform movements mostly of the upper extremity, accompanied by chewing and swallowing movements of the mouth. He also showed right-sided abdominal movements. Additional interesting features were the occurrence of an additional diplopia (objects appearing one above the other) after the initial diplopia had disappeared, in which the objects appeared from side to side. Again an Oppenheim and Gordon reflex appeared four weeks after the onset of the disease and also a distinct line of hyperalgesia a little below the mammary line in front and about the seventh cervical behind, together with a most distressing burning sensation in the fingers of the right hand, limited to the ulnar distribution. A Sergent's white line was persistent, showing a hypo-adrenia.

Dr. Hunt said that hiccup had not been a feature of the clinical symptoms observed by him. Abdominal waves were present, but the diaphragm was not affected. There was no evidence of organic disease of the brain other than delirium. He had, however, observed combination forms of epidemic encephalitis and myoclonus multiplex.

MEMORIAL RESOLUTIONS ON THE DEATH OF DR. E. E. SOUTHARD. J. Ramsay Hunt.

Mr. President: It is fitting that the members of this Society should pause in their deliberations and give expression to the loss we have sustained in the death of Dr. Southard. The news of his fatal illness in this city on February 8 touched us with a peculiar poignancy, as it came only a few days after his visit here as our guest. On that occasion he gave us one of those delightful evenings for which he was so justly famous, in which the subject of psychiatry was enlivened by his rare wit and philosophical mind. The memory of this occasion will always be treasured by those of us who heard him, for he was in one of his happiest moods. There are, I venture to say, but few other men in American medicine today who combine all the intellectual and personal gifts which made that address possible.

Elmer Ernest Southard was born in Boston in 1876, and at the time of his death was 43 years of age. He was a graduate of Harvard and of the Harvard Medical School, and subsequently became closely identified with his alma mater as Bullard Professor of Neuropathology and Assistant Professor of Psychology. At first a neuropathologist, he soon manifested an interest in psychiatry, and in 1912 became director of the Boston Psychopathic Hospital. This institution, under his leadership, grew to be one of the great centers for students of psychiatry in this country and Southard himself became a figure of national importance. He was an active and many-sided man. He held the directorship of the Eugenics Record office at Cold Spring Harbor, and was also a scientific director of the Psychological Laboratory at Bedford Hills. It was not, however, this array of appointments and honors that made Southard a unique and growing force in medicine, but rather his personality and philosophical type of mind.

A disciple of William James and Josiah Royce, he brought to medicine a mind which had been steeped and trained in the philosophical method. This, with his other gifts, made him one of the leaders of his generation.

While in his later years he became a man of affairs and an organizer of repute, he never lost the simple ideals and habits of the scholar, and this was perhaps one of his strongest characteristics, and contributed so largely to his unique position in medicine.

One can measure, in a way, what Southard had already accomplished in his life's work, but like all great men he was in the making and who can foretell what large accomplishments lay ahead of him? If one contemplates for a moment the loss to medicine if Sir William Osler had been struck down in early manhood while still teaching the Institutes of Medicine and McGill University, one can better gage what American psychiatry has lost with the passing of E. E. Southard.

TWO CASES OF BRAIN TUMOR. WITH SPECIMENS AND LANTERN SLIDE DEMONSTRATION. Presented by Drs. C. C. Beling, H. W. Martland (by invitation) and W. B. Eagleton (by invitation).

Dr. Beling: Case 1.—A man aged 25, an experimental engineer, suffered superficial burns of both corneas in an explosion of barium chlorate in 1913. Recovery was complete. In October, 1918, he began to see double, and lenses and general treatment failed to produce improvement. In March, 1919, an examination by Dr. Eagleton showed: vision in the right eye 20/100, in the left eye 20/50; marked papillitis of the right optic nerve; diplopia as a result of paralysis of the superior rectus of the right eye and a spontaneous nystagmus. There was increasing difficulty in looking upward. When Dr. Beling examined the patient the papillitis of the right optic nerve was marked. There was no deviation of the tongue or tremor. Knee jerks and plantar reflexes were normal except for a slight tendency to slow reaction on the right side in the latter. On May 15 the patient developed influenza and has been ill since that time. Since July 1 a dull pain on the top of his head with slight frontal headaches has persisted. His mental condition has apparently deteriorated. He was often nauseated and vomited. He could walk for a short time, then his body would stiffen and his head jerk back. An operation, a left subtemporal decompression, was performed by Dr. Eagleton. The brain was under great tension, the dura widely exposed. The operation was followed by an uninterrupted recovery, and the man's condition improved, although the papilledema persisted. Several weeks later, however, greatly increased intracranial pressure was apparent. For the first time he showed a tendency to fall backward. Examination at this time showed an intense double papilledema. There was generalized tremor and profuse hyperhidrosis and tonic contraction of the muscles. Knee reflexes were exaggerated. Clonus of the toes was found, the mental state was somewhat confused; the patient could not remember dates, but knew the year and that he was in the hospital. Cerebration was difficult and tremor was produced by attempts to answer questions. Priapism was noted; no abnormal psychosexual phenomena were present. His condition became steadily worse, the tremor increased, eyes bulged, jaws were set. On November 8 he began to have convulsions in rapid succession, profuse perspiration, and began to grow cyanotic. He died a few hours later.

Slides of the hospital history and the necropsy findings were shown by Dr. Martland. The diagnosis had been tumor of the midbrain. It was found, however, that there was a small psammoma of the pineal gland. An enormous dilatation of the third ventricle had resulted. The pineal gland was visible in the roentgenogram. Dr. Eagleton, in discussing the surgical features of this case, pointed out how useless further decompression would have been. The possibility of a pineal gland tumor had never been suggested, since the usual headache was lacking. Relief for the increased intracranial pressure was sought by the left sided decompression earlier in the case, since Dr. Eagleton had come to the conclusion that the patient was left handed; but this had had no effect on the papilledema.

Case 2.—A man aged 40 years, began to lose weight, vomited every morning, had increasing dizziness and began to see double. There were severe headaches. Examination by Dr. Beling showed slight swelling of the right papilla, vertigo, ataxia and asynergia of the right side. There was deviation to the right on walking, and nystagmus with rapid movements to the left and slow to the right. Hearing was about equal. Symptoms pointed to a lesion in the posterior chamber and subtentorial pressure. There was probably a cerebellar tumor with slight involvement of the pons since the left side of the face and hearing showed slight disturbance.

Dr. Eagleton noted the following phenomena: Romberg sign; spontaneous pointing deviation of the right hand to the right; more marked spontaneous nystagmus on looking to the right. Rotation to the right and sometimes to the left produced nystagmus, though whether spontaneous or induced could not be determined. Duration apparently was about eighteen seconds. Rotation to the left produced nystagmus of fifteen seconds' duration. There was deviation of both hands to the left. No dizziness appeared in either rotation. Cold caloric stimulation in the ears produced no nystagmus, no past pointing or dizziness. Nystagmus could be induced by turning the head backward, showing that tracts of the vertical canals were not functioning while the horizontal were functioning.

The tumor was thought to be pressing somewhat on both sides. Cases of this sort, Dr. Eagleton felt, where the exact nature of the tumor was not recognized, were better not operated on.

DISCUSSION

DR. J. W. STEPHENSON said that in his opinion the primary operation for any brain tumor should be puncture of the corpus callosum, especially puncture for midbrain tumors with which dilatation of the ventricles is most probable. Where large quantities of fluid are evacuated he advocated no further operation. In the absence of evacuation of fluid decompression was indicated. In tumors in locations other than the midbrain he would advise therapeutic lumbar puncture, particularly indicated in frontal tumors. He cited three cases of frontal tumors relieved of symptoms by lumbar puncture. In time the symptoms reappeared but lumbar puncture again relieved. In one case seven punctures were performed at intervals of seven to ten days, the puncture being dependent on return of symptoms. Each puncture gave decided relief.

DR. ALFRED TAYLOR advised wide removal of bone and the extensive opening of the dura in decompression cases, especially when the operation was on the left side as the speech center is more likely to be affected by limited work over it. In cases of high pressure ventricles should be aspirated. A needle inserted into the ventricle to evacuate the fluid was an assistance before opening the dura.

DR. E. D. FRIEDMAN mentioned the fact that the first patient showed an inability to rotate the eyes upward. This sign is very suggestive of lesions in the midbrain, since the more cephalad portion of the posterior longitudinal bundle controls the vertical movement of the eyes. This sign has been present in many of the cases of encephalitis seen in the present epidemic. Here, too, the peri-aqueductal localization of the encephalitis process was shown by this inability to rotate the eyes upward, together with oculomotor paralyses. Since this patient had already reached maturity, symptoms from involvement of the pineal body were absent. In younger persons lesions in this area involving the pineal gland are associated with sexual precosity and the premature development of the secondary sex characters. In one patient there were noted, with the other evidences of a lesion in this area, peculiar convulsive seizures, with athetoid movements in the hand, without loss of consciousness. This was attributed to involvement of the nucleus ruber which lies in the tegmentum of the brain stem.

Dr. Friedman asked, with reference to the second case, whether there was any conneal anesthesia on the side of the tumor. Oppenheim mentions this sign as being extremely diagnostic of posterior fossa neoplasm. It is homolateral to the tumor and said to be due to lesions of the spinal fibers of the trigeminus which in their ascent lie near the periphery.

DR. EAGLETON agreed with the advisability of wide opening of the dura for decompression when the case demanded, but emphasized again the usual danger to the speech center, and the uselessness of decompression in cases like the first, or cases that were as far advanced as the second.

TRAUMA AND OTHER NONSYPHILITIC INFLUENCES IN PARESIS.

Presented by Dr. Michael Osnato.

Literature yielded practically nothing on the influence of trauma in the production or precipitation of neurosyphilis. From the few cases which could be included under this category at the Vanderbilt Clinic in the past three or four years, from Dr. Osnato's own files only thirteen cases could be assembled. These cases were all proved either by laboratory examinations or necropsy. This deficiency of cases in which trauma was an associated factor, whether recognized or not, is to be noted in Southard and Solomon "Case History Series" in which a few cases only are mentioned. The post-traumatic paresis usually occurs, these authors state, citing Mott's study of the same subject, after at least a week's interval, since the time required for the destruction in the brain productive of the necessary symptoms would seem to be at least that long. Three months was the limit of time that Southard felt should be set to determine the influence of trauma as a causative factor. An increase in the number of cases of neurosyphilis during the war noted by the Canadian medical officers is thought to have been due to the great strain at the front, and the frequent physical injuries resultant on being buried.

A possible influence in the production or stimulation of paresis is accordingly granted by other observers. Dr. Osnato described the following cases in support of the traumatic theory: A patient who had been struck on the back of the head eighteen months before by a heavy object soon developed mental inefficiency. The only mental signs at present are perseveration of thought and speech and memory defect. The physical signs of paresis are present. A second patient, in whom the trauma was emotional, had been entirely efficient and dependable in his work until he was drafted into the army. His mental reaction was like a war neurosis in every respect. After diagnosis as a psychoneurotic and after his discharge he continued to fail and finally came to the clinic complaining of gross memory defects, fifteen-minute attacks of amnesia, dulness and retardation amounting almost to negativism and loss of interest. The mental picture was that of a psychoneurosis of the phobic type; a diagnosis of general paresis was made from investigation of the blood and spinal fluid. The problem of the emotional factors in the production of this condition is forcibly introduced here. The study must be speculative since the exact physiologic changes that may take place as a result of fear or other emotions is not known. A third patient developed the paretic picture after a prolonged etherization. Following an operation she complained of pains in the chest, legs, abdomen and right upper extremity. Grave memory defects also appeared. The physical signs of tabes were present, but mentally the patient was a general paretic. She is now under treatment and shows progressive mental deterioration, however, without delusions or hallucinations. The fourth patient had a severe attack of influenza and complained of lancinating pains in the right arm and both legs shortly after. She became depressed, slept badly, had tremor of the face, hands and tongue, was ataxic and had a moderate memory defect. The blood Wassermann reaction and spinal fluid findings in this case were those of a cerebrospinal syphilis rather than general paresis. Before the attack of influenza she had been perfectly well. In the last patient cited the trauma had been caused by a falling plank which struck the right parietal skull and glanced off striking the dorsal region of the spine. He was in Bellevue Hospital three days. There was evidence of a depressed fracture of the right vault of the skull in the frontoparietal region, over the Rolandic area. Left hemiplegia had developed when he left the hospital. A few days later there was unsteadiness of gait, ataxia, Romberg, typical paretic speech, stuttering, memory defect and tremor. The initial hemiplegia was undoubtedly due to the trauma. Up to the date of his injury he had worked steadily, and had shown no apparent signs of paresis.

Dr. Osnato emphasized that there are undoubted acute and chronic pathologic lesions of the brain ascribable to trauma of the head. Something seems to alter the permeability of the blood vessels of the brain, thus making possible the attack of the spirochetes on the brain tissue. In the cases described craniocerebral injury seems to have precipitated paresis. The toxins of influenza, infections or ether may have an effect similar to trauma, while the effect of emotional stress offers material for interesting investigation.

Dr. S. R. Leahy recalled Dr. Kraepelin's statement that paretic symptoms make their appearance immediately or shortly after a head injury. Sometimes the accident results from the paretic unsteadiness or occurs during a seizure. Whether in head injury, as is often assumed, one has to reckon with a circumstance which favors the outbreak of paresis, cannot, in the present state of our knowledge, be either proved or argued. Macfie Campbell, in his extensive works on paresis, states that even microscopically one cannot always tell which lesions have resulted from injury and which from paresis. Until we have more pathologic evidence the questions of the influence of injury in paresis must remain unsettled.

Dr. Osnato again pointed out that the patients had been efficient before injury, and then developed general paresis. Aside from the acute signs of trauma in the brain, other late signs, such as gliosis, nerve cell sclerosis, which recalled specifically the paretic brains, were to be found on necropsy. The same findings might be noted in traumatic insanity.

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